New RCEM curriculum revision notes

Covers all clinical SLOs from the curriculum.

Remember that the exams also contain questions from other SLOs such as research.

Items not individually referenced, life is too short. Some might say that life is too short for compiling a 1500 page revision document. They would be correct. Anyway, general references are at the end.

Mistakes are probably mine, or else transcribed from source. Information correct during 2021.

Enjoy/don't enjoy (delete as appropriate).

Good luck with exams.

RESUS

RP1 acute airway obstruction (17)

RP2 anaphylaxis/anaphylactoid reaction (20)

RP3 cardiorespiratory arrest (23)

RP4 major trauma (27)

RP5 respiratory failure (31)

RP6 sepsis (36)

RP7 shock (41)

RP8 unconsciousness (45)

RC1 choking (49)

RC2 stridor (49)

RC3 organ donation (53)

RC4 BRUE (55)

RC5 SUDIC protocol (58)

ALLERGY

AP1 acute allergy (62)

AP2 anaphylactoid reactions (63)

AP3 angioedema (66)

AP4 urticaria (68)

AC1 drug allergy/severe adverse drug reactions (71)

CARDIOLOGY

CP1 chest pain (76)

CP2 breathlessness (78)

CP3 palpitations (80)

CP4 transient loss of consciousness (84)

CC1 acute coronary syndromes (90)

CC2 myocardial infarction (97)

CC3 arrhythmias (105)

CC4 cardiac failure (112)

CC5 cardiac tamponade (116)

CC6 congenital heart disease (121)

CC7 diseases of the arteries, including aortic dissection (131)

CC8 diseases of myocardium (146)

CC9 hypertensive emergencies (154)

CC10 pacemaker function and failure (157)

CC11 pericardial disease (161)

CC12 sudden cardiac death (167)

CC13 valvular heart disease (176)

DERMATOLOGY

DP1 dermatological manifestations of systemic illness (191)

DP2 rashes (193)

DC1 common childhood exanthems (197)

DC2 cutaneous drug reactions (212)

DC3 eczema (217)

DC4 erythroderma (220)

DC5 infections of the skin and soft tissues (222)

DC6 necrotising fasciitis (238)

DC7 pressure ulcers (241)

DC8 purpuric rash including Henoch Schönlein purpura (244)

DC9 Stevens-Johnson syndrome (249)

DC10 toxic epidermal necrolysis (253)

DC11 urticaria (257)

EAR, NOSE AND THROAT

EP1 ENT foreign bodies (260)

EP2 ENT injuries (264)

EP3 epistaxis (268)

EP4 hearing loss (273)

EP5 painful ear (276)

EP6 sore throat (278)

EP7 vertigo (281)

EC1 croup (287)

EC2 epiglottitis (293)

EC3 glandular fever (294)

EC4 LMN facial nerve palsy (295)

EC5 Ménière's disease (297)

EC6 nasal fractures (297)

EC7 otitis externa (298)

EC8 otitis media (300)

EC9 pharyngitis (301)

EC10 post-tonsillectomy bleed (302)

EC11 tonsillitis (303)

EC12 tracheostomy emergencies (304)

EC13 quinsy (307)

EC14 salivary gland disease (308)

EC15 vestibular neuritis (314)

ELDERLY CARE/FRAILTY

ElP1 delirium (316)

ElP2 deterioration in mobility (320)

ElP3 falls (323)

ElP4 fragility fractures (328)

ElP5 frailty (332)

ElP6 hypothermia (333)

ElP7 incontinence (335)

ElP8 increasing care needs (339)

ElP9 memory loss (342)

ElP10 unsteadiness/balance disturbance (345)

ElC1 comprehensive geriatric assessment (349)

ElC2 acute confusion (351)

ElC3 ceiling of care and end of life care (356)

ElC4 dementia – cognitive impairment (358)

ElC5 fragility fractures (362)

ElC6 mobility (365)

ElC7 osteoporosis (368)

ElC8 pharmacology considerations in the older patient (370)

ENDOCRINOLOGY

EnP1 Addisonian crisis (373)

EnP2 hyperglycaemia (377)

EnP3 hypoglycaemia (378)

EnC1 adrenal disorders (380)

EnC2 diabetic ketoacidosis (385)

EnC3 diabetes mellitus and complications including diabetic foot (387)

EnC4 hyperosmolar hyperglycaemic state (400)

EnC5 pituitary disorders (404)

EnC6 thyroid emergencies (413)

ENVIRONMENTAL EMERGENCIES

EnvC1 heat stroke and heat exhaustion (425)

EnvC2 drug-related hyperthermias (427)

EnvC3 hypothermia and frostbite (431)

EnvC4 decompression sickness (434)

EnvC5 near-drowning (437)

EnvC6 radiation exposure and safety (439)

EnvC7 industrial chemical incidents (443)

EnvC8 bites and envenomations typical for the UK (445)

EnvC9 high altitude emergencies – cerebral and pulmonary oedema (446)

EnvC10 acid attacks (448)

GASTROENTEROLOGY AND HEPATOLOGY

GP1 abdominal and loin pain (449)

GP2 abdominal swelling or mass (452)

GP3 ascites (454)

GP4 constipation (460)

GP5 diarrhoea (462)

GP6 haematemesis and melaena (463)

GP7 jaundice (464)

GP8 anal pain and rectal bleeding (468)

GP9 nausea and vomiting (470)

GP10 dysphagia (472)

GC1 alcohol related liver disease including withdrawal (475)

GC2 decompensated cirrhosis (485)

GC3 dehydration in children (491)

GC4 functional bowel disorders (495)

GC5 gastrointestinal infections (504)

GC6 hepatitis (508)

GC7 inflammatory bowel disease (518)

GC8 peptic ulcer disease (523)

GC9 pyloric stenosis (527)

HAEMATOLOGY

HP1 anaemia (529)

HP2 bruising and spontaneous bleeding (532)

HP3 massive haemorrhage (543)

HC1 anti-coagulant reversal (547)

HC2 DIC (548)

HC3 haemophilia (549)

HC4 ITP (552)

HC5 leukaemia (553)

HC6 lymphoma (557)

HC7 marrow failure (561)

HC8 sickle cell disease/crisis (562)

HC9 transfusion reactions (563)

INFECTIOUS DISEASES

IP1 fever (565)

IP2 pyrexia in travellers (567)

IP3 sepsis (569)

IP4 needlestick injury/exposure to blood borne viruses (571)

IC1 influenza (577)

IC2 infection in immunocompromised patients (579)

IC3 infestations (581)

IC4 Kawasaki disease (588)

IC5 notifiable diseases (590)

IC6 pyrexia of unknown origin – different age groups (591)

IC7 malaria (594)

IC8 HIV infection (596)

MAXILLOFACIAL/DENTAL

MaP1 dental pain (598)

MaP2 facial swelling (599)

MaP3 avulsed or fractured teeth (600)

MaP4 facial bone injury (603)

MaC1 dental abscess (605)

MaC2 facial wounds (607)

MaC3 post extraction complications (610)

MaC4 TMJ dislocation (611)

MENTAL HEALTH

MHP1 aggressive or disturbed behaviour (613)

MHP2 anxiety/panic (613)

MHP3 physical symptoms unexplained by organic disease (623)

MHP4 self-harm (624)

MHP5 refusal of treatment (625)

MHC1 alcohol and substance misuse (627)

MHC2 depression (631)

MHC3 eating disorders (634)

MHC4 personality disorders (642)

MHC5 acute psychosis including bipolar, schizophrenia (648)

MHC6 somatic symptom disorders (659)

MHC7 stress disorders (661)

MHC8 suicide (665)

MUSCULOSKELETAL (NON-TRAUMATIC)

MuP1 acute back pain (668)

MuP2 limb pain and swelling (675)

MuP3 neck pain (679)

MuP4 joint swelling (687)

MuP5 acute hot swollen joint (689)

MuC1 cauda equina syndrome (692)

MuC2 crystal related arthropathies (693)

MuC3 septic arthritis (699)

MuC4 limb pain and swelling: bursitis and tendonitis in the upper and lower limb including ruptured biceps, Achilles tendonitis, plantar fasciitis, metatarsalgia, carpal tunnel and other entrapment neuropathies plus sinister causes including bone tumour, stress fracture (702)

MuC5 spinal pain and radiculopathy (738)

MuC6 risks of rheumatological disease modifying drugs (744)

MuC7 spinal infections (746)

MuC8 torticollis (749)

MuC9 limping child (750)

MuC10 osteochondritis (759)

NEONATAL EMERGENCIES

NeoC1 delivery and resuscitation of the newborn (762)

NeoC2 neonatal sepsis (767)

NeoC3 cyanotic/non-cyanotic congenital heart disease (770)

NeoC4 jaundice (776)

NeoC5 feeding patterns (778)

NEPHROLOGY

NepP1 electrolyte disorders (782)

NepP2 oliguria (787)

NepC1 acute kidney injury (789)

NepC2 drugs and the kidney (794)

NepC3 electrolyte disorders (798)

NepC4 fluid balance disorders (809)

NepC5 renal replacement therapy (814)

NEUROLOGY

NeuP1 acute confusion (818)

NeuP2 headache (823)

NeuP3 seizures/status epilepticus (836)

NeuP4 speech disturbance (845)

NeuP5 hemiparesis/hemiplegia (848)

NeuP6 gait abnormality (850)

NeuP7 visual disturbance (852)

NeuP8 weakness/paralysis (859)

NeuP9 dizziness and vertigo (864)

NeuC1 botulism (872)

NeuC2 cerebral venous sinus thrombosis (873)

NeuC3 febrile convulsion (876)

NeuC4 functional illness (877)

NeuC5 Guillain-Barré (880)

NeuC6 meningitis and encephalitis (882)

NeuC7 multiple sclerosis (885)

NeuC8 myaesthenia gravis (888)

NeuC9 Parkinson's disease and other movement disorders (889)

NeuC10 peripheral neuropathy (acute) (894)

NeuC11 subarachnoid haemorrhage (895)

NeuC12 stroke and TIA (898)

NeuC13 tetanus (910)

NeuC14 tumours involving the brain and spinal cord (915)

NeuC15 VP shunts (927)

NeuC16 Wernicke's encephalopathy (932)

OBSTETRICS AND GYNAECOLOGY

ObP1 pelvic pain (935)

ObP2 vaginal bleeding (938)

ObP3 pregnancy (944)

ObP4 genital injury/assault (948)

ObP5 vaginal discharge (952)

ObP6 foreign bodies (955)

ObP7 patient in labour (956)

ObC1 ante-partum haemorrhage (962)

ObC2 bleeding in early pregnancy (963)

ObC3 exposure to infections during pregnancy e.g. chickenpox (965)

ObC4 ectopic pregnancy (972)

ObC5 genital injury/Female Genital Mutilation (973)

ObC6 HELLP (974)

ObC7 heavy menstrual bleeding (976)

ObC8 hyperemesis gravidarum (980)

ObC9 maternal collapse (981)

ObC10 post-partum haemorrhage (983)

ObC11 pre-eclampsia/eclampsia (984)

ObC12 pelvic infection (986)

ObC13 post menopausal bleeding (988)

ObC14 prescribing in pregnancy (988)

ObC15 Rhesus D prophylaxis (993)

ObC16 sepsis in and following pregnancy (993)

ObC17 thrombosis during and following pregnancy (998)

ObC18 trauma in pregnancy (1000)

ObC19 OHSS (1004)

ONCOLOGICAL EMERGENCIES

OncP1 acute presentations of undiagnosed cancer that may present to the ED (including weight loss, dysphagia, pain etc.) (1007)

OncC1 complications related to local tumour progression e.g. acute cord compression, upper airway obstruction, pericardial and pleural effusions, SVC compression syndrome, raised intracranial pressure (1008)

OncC2 complications relating to cancer treatment including neutropaenic sepsis, anaemia and thrombocytopaenia, and immunotherapy (1017)

OncC3 biochemical complications of malignancy – hypercalcaemia, SIADH, adrenocortical insufficiency (1030)

OPHTHALMOLOGY

OptP1 diplopia (1034)

OptP2 eye trauma including foreign bodies (1036)

OptP3 painful eye (1037)

OptP4 red eye (1037)

OptP5 sudden visual loss (1038)

OptC1 acute glaucoma (1041)

OptC2 cranial nerve palsy (1042)

OptC3 orbital/preseptal and peri-orbital cellulitis (1049)

OptC4 ophthalmia neonatorum (1051)

OptC5 inflammatory eye disease (1052)

OptC6 temporal arteritis (1055)

PAIN AND SEDATION

PC1 analgesics (1055)

PC2 non-pharmacological methods of pain management (1059)

PC3 pain assessment (1060)

PC4 sedation (1064)

PALLIATIVE AND END OF LIFE CARE

PalP1 advanced malignancy and end stage chronic disease (1074)

PalC1 advanced care planning (1076)

PalC2 anticipatory medications (1079)

PalC3 end stage organ failure (1078)

PalC4 pain management (1088)

PalC5 physical symptoms other than pain (1092)

PalC6 psychosocial concerns including spiritual care and care of the family (1101)

PalC7 the dying patient (1102)

PHARMACOLOGY AND POISONING

PhP1 medication side effects/interactions (1106)

PhP2 overdose (1111)

PhP3 accidental poisoning (1117)

PhC1 overdose of prescription and non-prescription medications including legal and non-legal drugs (1123)

PhC2 poisoning – carbon monoxide, cyanide, organophosphate (1142)

PhC3 toxidromes (1148)

PhC4 use of antidotes (1151)

PhC5 batteries, household chemicals, poisonous plants (1154)

RESPIRATORY

RP1 chest pain (1159)

RP2 breathlessness (1160)

RP3 haemoptysis (1164)

RP4 cough (1166)

RC1 asthma (1170)

RC2 bronchiolitis (1179)

RC3 COPD (1182)

RC4 foreign body inhalation (1188)

RC5 pertussis (1189)

RC6 pleural effusion (1191)

RC7 pneumonia (1199)

RC8 pneumothorax (1209)

RC9 pulmonary aspiration (1215)

RC10 pulmonary embolus (1217)

RC11 viral induced wheeze in children (1225)

SEXUAL HEALTH

SeP1 genital discharge (1226)

SeP2 genital lesions (1232)

SeP3 emergency contraception (1237)

SeP4 post-exposure prophylaxis (1243)

SeC2 sexual assault (1246)

SeC3 sexually transmitted infections (1248)

SURGICAL EMERGENCIES

SuP1 abdominal pain (1258)

SuP2 abdominal swelling/mass (1262)

SuP3 constipation (1262)

SuP4 diarrhoea (1271)

SuP5 GI bleeding (1279)

SuP6 anal/rectal pain (1287)

SuP7 nausea/vomiting (1291)

SuC1 ano-rectal abscesses (1296)

SuC2 appendicitis (1298)

SuC3 biliary colic (1302)

SuC4 bowel obstruction (1304)

SuC5 breast abscess (1308)

SuC6 cholangitis (1311)

SuC7 cholecystitis (1313)

SuC8 diverticular disease (1314)

SuC9 haemorrhoid disease (1318)

SuC10 hernias (1319)

SuC11 intussusception (1321)

SuC12 ischaemic bowel (1325)

SuC13 lower gastrointestinal and rectal bleeding (1329)

SuC14 pancreatitis (1322)

SuC15 viscus perforation (1336)

SuC16 volvulus (1338)

TRAUMA

TP1 head injury (1340)

TP2 spinal injury (1347)

TP3 chest and lung injury (1356)

TP4 major vascular injury (1365)

TP5 abdominal injury (1369)

TP6 pelvic injury (1375)

TP7 limb and joint injury (1382)

TP8 burns (1387)

TP9 inhalational injury (1396)

TP10 wounds (1399)

TC1 compartment syndrome (1406)

TC2 limb and joint injury including bony, musculo-tendinous and complications (1407)

TC3 electrical burns (1430)

TC4 Salter-Harris classification (1432)

TC5 infection – paronychia, pulp space, flexor sheath, nail bed, amputations etc. (1435)

TC6 animal bites including human (1445)

TC7 injury to bladder, urethra, testes or penis (1451)

UROLOGY

UP1 dysuria (1456)

UP2 injury to bladder, urethra, testes or penis (1458)

UP3 urinary retention (1462)

UP4 testicular pain/swelling (1468)

UP5 loin pain (1472)

UP6 haematuria (1476)

UC1 epididymo-orchitis (1480)

UC2 renal stone disease (1482)

UC3 phimosis/paraphimosis (1486)

UC4 priapism (1489)

UC5 testicular torsion (1492)

UC6 prostatitis (1493)

UC7 UTI/pyelonephritis (1495)

VASCULAR

VC1 acute limb ischaemia (1498)

VC2 aortic aneurysmal disease (1504)

VC3 DVT (1507)

OTHER CLINICAL PRESENTATIONS

XC1 major incident management (1513)

XC2 PHEM (1519)

XC3 safeguarding in adults (1528)

XC4 domestic abuse (1530)

SAFEGUARDING AND PSYCHO-SOCIAL EMERGENCIES IN CHILDREN

SaP1 self-harm in children and adolescents (1533)

SaP2 concerning presentation (1535)

SaC1 conditions presenting as a symptom of NAI or psychological distress e.g. deliberate self harm, aggression or risk taking behaviour, recurrent abdominal pain, headaches or faints, recurrent attendances in young children (1540)

SaC2 roles of other systems in protecting children e.g. Social Services, the Child Protection Plan, Police Child Protection and Domestic Violence Units, SureStart, Childline, Health Visitors, School Nurses (1554)

SaC3 Mental illness in childhood including depression, anxiety, OCD, bipolar and schizophrenia (1556)

SaC4 sexual abuse (1563)

RESUS

RP1 acute airway obstruction

- types
    - incomplete
        - partial upper airway obstruction
        - ability to breathe maintained
        - inspiratory stridor and increased work of breathing
    - complete
        - inability to talk, cough or breathe
        - apnoea and cyanosis
        - paradoxical breathing may be present
    - locations
        - luminal
            - e.g. foreign body
        - intramural
            - e.g. tumour, neuromuscular disease
        - extramural
            - e.g. thyroid mass
- causes
    - foreign body
    - infection
        - epiglottitis
        - retropharyngeal abscess
        - bacterial tracheitis
        - Ludwig's angina
        - laryngotracheitis
        - diphtheria
        - tetanus
    - immune
        - angioedema
        - anaphylaxis
    - tumour
    - trauma
        - neck haematoma
        - laryngeal fracture
        - burns
        - post-operative complications
    - poisoning and toxic exposures
        - smoke inhalation
        - caustic ingestion
        - strychnine poisoning
    - laryngospasm
        - physical or chemical stimuli
        - drug induced
            - e.g. acute dystonic reaction, ketamine

    - congenital
        - vascular rings
        - laryngeal webs
    - other
        - paradoxical movement of the vocal cords
        - altered level of consciousness
        - cranial nerve palsies
        - paralysis
        - hysterical stridor
        - myoedema
            - percussion or flicking of a muscle belly results in a non-tender swelling or ridge resolving in a few seconds
            - thought to be due to release of calcium ions on percussion and delayed reaccumulation in the sarcoplasmic reticulum
            - may be seen in hypothyroidism, hypoalbuminaemia, rabies, severe cachexia, hyponatraemia and renal failure
- history
    - progression of symptoms
    - positional exacerbation
    - whether patient wakes at night with difficulty in breathing
    - dysphagia
    - drooling
    - chest infections due to reduced ability to cough
- examination
    - stridor
        - at rest implies a reduction in airway diameter of >50%
    - airway assessment
    - neck examination
    - nasal endoscopy
        - does not involve local anaesthetic to the cords which could precipitate complete airway obstruction
- management
    - may include awake tracheostomy or inhalational induction
    - emergency front of neck access in respiratory arrest
    - awake tracheostomy
        - indications:
            - severe stridor
            - large tumour
            - gross anatomical distortion
            - larynx not visible on nasal endoscopy
        - technique
            - no sedation
            - Heliox improves symptoms
            - high concentrations of O2 can precipitate nitrogen narcosis
            - prepare in sitting position
- surgical cricothyroidotomy
    - indication

        - can’t intubate, can’t ventilate scenario
    - contraindications
        - ability to secure an airway with less invasive means
        - airway trauma rendering access via the cricothyroid membrane futile
            - e.g. laryngeal fracture, tracheal transection
            - requires tracheostomy or access via the traumatic opening
        - children <10 years
            - prone to laryngeal trauma and have a high incidence of post-operative complications
            - needle cricothyrotomy is advised (life-saving surgical cricothyroidotomy has successfully been performed on children)
    - approach
        - ideally in an anticipated difficult airway one person should attempt intubation whilst another prepares for FONA
            - locate cricothyroid membrane, mark and infiltrate skin and fascia with lidocaine with adrenaline (whilst patient awake if co-operative)
            - equipment open and ready to proceed if needed
        - sterile preparation may not be possible in a true emergency
        - consider sedation (e.g. 20mg IV ketamine); in a true emergency patient will become obtunded due to hypoxia
        - extend neck in supine position, palpate for cricothyroid membrane
            - stabilise thyroid cartilage with non-dominant hand
        - usually best to approach with vertical incision first
            - longer if anatomy distorted
        - dissect down with scalpel, fingers or forceps until membrane identifiable
        - horizontal incision through membrane then twist scalpel 180 degrees
        - pass bougie and remove scalpel
        - size 6.0 ET tube over bougie and into trachea
            - twist ETT as it passes through skin
            - advance only until balloon in trachea
        - remove bougie and connect ETT to BVM
        - confirm placement
    - complications of FONA
        - failure
        - bleeding
        - infection
        - damage to local structures
            - e.g. larynx, vessels, nerves, oesophagus, cartilage, muscle
        - cricoid fracture
        - fistula formation
        - scarring
        - hypoxia
        - death
- needle cricothyroidotomy
    - indications
        - can’t intubate, can’t oxygenate scenario

        - can buy time for definitive procedure but does not allow ventilation and leads to hypercapnia
    - contraindications
        - local infection
        - non-identifiable anatomy
            - e.g. severe obesity, trauma, swelling, mass lesion
        - previous failed attempts
    - procedure
        - extend neck and identify and stabilise cricothyroid membrane
        - hold a 5ml syringe with 1-2ml saline attached to a 14g cannula in the dominant hand
        - insert at 45 degrees in a caudal direction
        - aspirate whilst advancing, stop when air aspirated
            - end point is aspiration of air up the entire 5ml barrel
        - stabilise the cannula hub with the non-dominant hand and release the plunger of the syringe
            - it will stay in position if cannula correctly placed and be sucked back down if incorrectly placed
        - advance the cannula over the needle into the trachea and remove the needle
        - repeat full aspiration of air into 5ml syringe with 1-2ml saline
            - if initial aspiration fails withdraw cannula slightly in case it is against the posterior tracheal wall
        - attach appropriate oxygen supply
    - complications
        - failure
            - poor technique in stressful situation
            - kinking of cannula
            - blood or vomitus in airway
            - difficult anatomy
        - cannula obstruction
        - cannula dislodgement
        - injury to local structures
        - surgical emphysema (if high flow oxygen administered through malpositioned cannula

RP2 anaphylaxis/anaphylactoid reaction

- background
    - severe, life-threatening generalised or systemic hypersensitivity reaction
    - causes around 20 deaths/year in the UK
    - requires:
        - acute onset with sudden progression
        - skin or mucosal changes
        - life threatening ABC problems
- pathophysiology
    - IgE mediated hypersensitivity reaction to an antigen
        - immediate type 1 hypersensitivity

        - an antigen binds to an antigen-specific antibody
        - leads to histamine and serotonin release from basophil and mast cell degranulation
    - biphasic reactions
        - occur about 5% of the time
        - may occur >24 hours after the initial episode
        - usually less severe than the initial episode
        - possible risk factors include:
            - delayed administration of adrenaline
            - slow response to adrenaline
            - need for repeated doses of adrenaline
            - need for IV fluids
- clinical features
    - exposure to antigen
        - stings
        - foods
        - antibiotics
        - contrast media
        - thrombolytics
        - NSAIDs
        - suxamethonium
        - non-depolarising neuromuscular blockers
    - rapid onset (within minutes) of some combination of:
        - airway problems
            - lip and tongue swelling/angioedema
            - nasal congestion
            - sneezing
            - tightness of throat/hoarse voice/stridor
        - breathing problems
            - tachypnoea
            - bronchospasm/wheeze
            - increased mucous secretions
            - exhaustion
            - cyanosis
            - respiratory arrest
        - circulation problems
            - hypotension (may be persistent and can be the only sign)
            - tachycardia
            - arrhythmia
            - myocardial ischaemia
            - cardiac arrest
        - neurological problems
            - confusion
            - agitation
            - loss of consciousness
        - skin and mucosal problems (can be subtle or absent)
            - urticaria

            - erythema
            - pruritus
        - gastrointestinal
            - stomach cramps
            - nausea
            - vomiting
            - diarrhoea
        - other
            - feeling of impending doom
- investigations
    - serum tryptase
        - at 1, 6 and 24 hours
        - normal <1ng/ml
        - non-specific and anaphylactoid reactions 1-15ng/ml
        - true anaphylaxis >15ng/ml
    - skin testing
        - diagnostic for anaphylaxis but not anaphylactoid reactions
        - should take place 4-6 weeks post event to allow regeneration of IgE
        - antihistamines should not have been given in the past 5 days
        - all drugs given before the first event should be tested with saline used as a negative control and histamine solution as a positive control
        - a weal >2mm wider than saline is a positive result
        - repeat positive test with a 1:10 dilution to reduce chance of false positive
        - if positive drug detected, other drugs in the same class should be tested
        - if skin prick test is negative but history is strong, intra-dermal testing with diluted drugs can be done
- management
    - remove/stop trigger
    - supine position (head down)
    - oxygen
    - exclude alternative diagnoses
    - if pulse present:
        - adrenaline 0.5mg IM
        - IV access
        - fluid boluses if systolic BP <90 mmHg
        - chlorphenamine 10mg IV
        - hydrocortisone 200mg IV
            - may be useful for preventing biphasic response
            - steroids no longer recommended for discharge
        - repeat adrenaline after 5 minutes if persistent hypotension/bronchospasm
            - if still persisting after 5 minutes, start adrenaline infusion
    - if pulse absent:
        - CPR
        - raise legs
        - 2 large IVs
        - 2 litres of fluid
        - increasing adrenaline (1-4mg in adults, 10-100microgram/kg in children)

        - antihistamine
        - extended CPR
    - persisting hypotension
        - ranitidine (H2 antagonism)
        - adrenaline/noradrenaline infusion
        - 1mg (1ml of 1:1000) adrenaline in 100ml 0.9% sodium chloride
            - via infusion pump and dedicated line
        - on different side to BP cuff to reduce risk of interruption and extravasation
            - start at 0.5-1.0ml/kg/hr and titrate
        - invasive monitoring
        - consider colloid
    - persistent bronchospasm
        - treat as for asthmatic emergencies
    - persisting angioedema
        - nebulised adrenaline (1mg)
        - intubation
        - cricothyroidotomy
        - tracheostomy
    - C1 esterase inhibitor deficiency
        - resistant to steroids, adrenaline and antihistamines
        - requires C1 esterase inhibitor concentrate
        - if unavailable, 2 units of fresh frozen plasma is usually effective
- disposition
    - observe for a minimum of 6 hours in most cases
    - admit it:
        - severe anaphylaxis
        - uncontrolled asthma
        - slow response to adrenaline
        - need for bolus fluids
        - need for a second dose of adrenaline
        - possibility of continuing absorption (e.g. fully eaten substance)
        - poor access to emergency care
- discharge
    - appropriate discharge instructions (written action plan)
    - teach use of adrenaline auto-injector
    - provide prescriptions and ensure they can get them
    - ensure access to emergency services
    - arrange follow up (e.g. GP, medic alert bracelet, allergy clinic referral)
    - record allergy in clinical notes

RP3 cardiorespiratory arrest

- in-hospital resuscitation (Resus Council)
    - shout for help and assess patient
    - check for signs of life (consciousness, breathing, carotid pulse) – confirm arrest
        - high quality CPR
            - oxygen and airway adjuncts
            - switch compressor at every rhythm assessment

        - defibrillation
            - apply pads and turn on defibrillator
            - attempt defibrillation if indicated
        - ALS
            - when sufficient trained personnel present
- ALS
    - unresponsive and not breathing normally
        - maintain personal safety
        - CPR 30:2
        - attach defibrillator/monitor
        - high quality chest compressions and:
            - oxygen
            - waveform capnography
            - continuous compressions if advanced airway
            - minimise interruptions to compressions
            - IV or IO access
            - adrenaline every 3-5 minutes
            - amiodarone after 3 shocks
            - identify and treat reversible causes
        - shockable (VF/pulseless VT)
            - 1 shock
                - recommended level
                    - if unsure, use at least 120-150J to start
                    - use same or higher energy for subsequent shocks
            - immediately resume CPR for 2 minutes
            - drugs:
                - adrenaline
                    - cardiac arrest dose 1mg IV/IO
                    - give after third shock
                        - then every 3-5 minutes
                - amiodarone
                    - 300mg IV/IO
                    - give after third shock
                    - consider further 150mg IV/IO after 5 shocks
        - non-shockable (PEA/asystole)
            - CPR
            - drugs:
                - adrenaline
                    - cardiac arrest dose 1mg IV/IO
                    - give as soon as possible
                    - further doses every 3-5 minutes
        - reversible causes
            - hypoxia
            - hypovolaemia
            - hypo-/hyperkalaemia/metabolic
            - hypo-/hyperthermia
            - thrombosis

        - coronary or pulmonary
    - tension pneumothorax
    - tamponade
    - toxins
- consider
    - consider ultrasound to identify reversible causes
    - coronary angiography/percutaneous coronary intervention
    - mechanical chest compression to facilitate transfer/treatment
    - extracorporeal CPR
- ROSC
    - use an ABCDE approach
    - aim for SpO_2 94-98% and normal $PaCO_2$
    - 12 lead ECG
    - identify and treat cause
    - targeted temperature management
- ABCDE
    - airway
        - causes
            - CNS depression
            - blood
            - vomitus
            - foreign body (e.g. tooth, food)
            - direct trauma to face or throat
            - epiglottitis
            - pharyngeal swelling (e.g. infection, oedema)
            - laryngospasm
            - bronchospasm
            - bronchial secretions
            - blocked tracheostomy
        - recognition
            - look, listen, feel:
                - snoring
                - gurgling
                - stridor
                - wheeze
                - see-saw breathing
                - in complete obstruction, silence with no air movement
        - treatment
            - open airway with:
                - head tilt/chin lift or jaw thrust
                - OPA
                - supraglottic airway
                - tracheal intubation
            - use suction if required
            - give oxygen

- maintain normal sats (94-98% unless risk of hypercapnic respiratory failure, then 88-92%)

- breathing
  - causes
    - decreased respiratory drive
      - e.g. CNS depression
    - decreased respiratory movement
      - e.g. spinal cord injury, muscle weakness, rib injury
    - lung disorders
      - e.g. severe pneumonia, advanced chronic lung disease
  - recognition
    - shortness of breath and distress in conscious patient
    - symptoms of hypoxaemia/hypercapnia:
      - irritability
      - confusion
      - lethargy
      - decrease in conscious level
    - cyanosis (late sign)
    - increased respiratory rate indicates breathing problems
    - pulse oximetry and blood gas to assess adequacy of oxygenation and ventilation
  - treatment
    - treat underlying cause
    - oxygen to correct hypoxaemia
    - consider respiratory support
      - high flow nasal oxygen
      - continuous positive pressure ventilation (CPAP)
      - non-invasive ventilation (NIV)
      - sedation, tracheal intubation and controlled ventilation
- circulation
  - causes
    - primary heart problems
      - e.g. acute coronary syndrome
    - other problems
      - e.g. hypovolaemia, sepsis, hypoxia
  - recognition
    - signs and symptoms of cardiac disease
      - chest pain
      - shortness of breath
      - syncope
      - tachycardia

                - bradycardia
                - cardiac arrest
                - tachypnoea
                - hypotension
                - poor peripheral perfusion
                - altered mental state
                - oliguria
        - treatment
            - treat underlying cause
                - ACS
                    - aspirin
                    - nitrates
                    - opioids
                    - oxygen if required
                - stop bleeding and give blood
- disability
    - causes of unconsciousness
        - profound hypoxia
        - hypercapnia
        - cerebral hypoperfusion
        - sedatives/analgesics
    - treatment
        - review and treat ABCs
        - check for drug included causes
            - consider naloxone
        - examine pupils
        - rapid assessment of conscious level using ACVPU
            - **a**lert
            - **n**ew confusion
            - **v**ocal stimuli
            - **p**ainful stimuli
            - **u**nresponsive
        - alternatively use GCS
        - measure blood glucose and correct hypoglycaemia
        - nurse in lateral position if airway not protected
- exposure
    - full exposure
        - respect for dignity
        - minimisation of heat loss
        - look for rashes and injuries

RP4 major trauma

- definition
    - major injury affecting more than one body system
    - Injury Severity Score >15
- trauma call criteria
    - varies between hospitals

    - criteria usually combine mechanism, specific injuries, physiological derangement, patient factors, physician discretion
    - factors likely to trigger a trauma call include:
        - mechanism
            - falls >6m
            - high risk motor vehicle collisions
            - vehicle versus cyclist
            - vehicle versus pedestrian
        - specific injuries
            - flail chest
            - paralysis
            - proximal penetrating injuries or amputations
            - multiple long bone fractures
            - pelvic fractures
            - crushed or mangled extremity
        - physiological derangement
            - GCS <14
            - SBP <90mmHg
            - RR >30 or <10
        - patient factors
            - extremes of age
            - pregnancy
            - bleeding diathesis
- key steps in trauma management
    - preparation, triage and activation of the trauma team
        - transfer from trolley to resus bed using spinal precautions if indicated
        - obtain handover from the prehospital care providers including history, mechanism of injury, prehospital treatment, response to treatment
        - obtain a set of observations
    - primary survey (ABCDE) and resuscitation
        - as needed
        - large bore IV access
        - bloods for G&S/crossmatch and baseline bloods
    - adjuncts to primary survey and resuscitation
        - catheter
        - NG tube
        - ECG monitoring
        - bedside ultrasound
        - portable x-ray
    - consider need for patient transfer
        - as soon as adequate information available
    - secondary survey
    - adjuncts to the secondary survey
        - further imaging and investigations
    - continued post-resuscitation monitoring and re-evaluation
    - definitive care and disposition
- preparation

- people
    - consider trauma team activation
    - ensure continued safe running of the rest of the emergency department
    - support for family if needed
    - security if needed (e.g. gunshot or knife wound where there may be risk of assailant attending)
    - notify other hospital areas as required (e.g. labs, radiology, theatres, ICU)
- place
    - ideally designated trauma bay with facilities for resuscitation
- equipment and drugs
    - anticipate what might be needed using A-E approach
    - potential requirements include:
        - universal precautions
        - advanced airway equipment
        - analgesia
        - RSI drugs
        - chest tubes
        - rapid infuser
        - activation of massive haemorrhage protocol
        - pelvic binders
        - femoral splints
        - warming equipment
        - emergency thoracotomy tray

- trauma team roles
    - varies by size of centre and may be just a doctor and nurse in smaller centres
    - important aspects are:
        - team members are trained in trauma care
        - clear roles and organisation
        - effective communication
        - support from other hospital areas, transfer areas and trauma centres
    - team roles include:
        - team leader
            - usually senior emergency medicine doctor
            - co-ordinates assessment and management
        - airway doctor
            - doctor with advanced airway skills (ED, anaesthetics or ICU)
        - procedures doctor
            - emergency doctor
        - assessment doctor
            - emergency or surgical doctor
        - airway nurse
            - nurse trained to assist with advanced airway management
        - drugs/procedures nurse
        - scribe
            - nurse who keeps a real time written record of events and interventions
        - runners

                - healthcare assistants who relay messages, source equipment and assist in transfers
        - other speciality doctors may be alerted as necessary (e.g. ENT, ophthalmology)
- morbidity and mortality from major trauma
    - leading cause of death in under 40s in developed countries
        - also a major killer of older age groups
    - most victims are young males
    - there is massive additional societal burden for survivors from morbidity
    - most preventable deaths are due to haemorrhage
    - pattern of death from major trauma
        - immediate
            - seconds to minutes after injury
            - usually unpreventable
                - e.g apnoea due to high spinal or brain injury, catastrophic haemorrhage due to great vessel disruption
        - early
            - minutes to hours after injury
            - usually haemorrhage related
            - ATLS style emergency care specifically targets these patients
        - late
            - days to weeks after injury
            - usually due to multi-organ failure or sepsis
            - optimal early management may prevent these
- <c>ABCDE considerations
    - catastrophic haemorrhage
        - immediate pressure on wound
        - tourniquet may be required
    - airway
        - assess and apply jaw thrust if needed
        - c-spine immobilisation with manual inline stabilisation if indicated
    - breathing
        - check for breathing
        - check respiratory rate and saturations
        - high flow oxygen during initial assessment
        - sucking chest wounds
            - cover with gauze dressing
        - equal chest wall movement
            - if suspicion of tension pneumothorax, perform needle decompression or portable CXR if stable
            - consider massive haemothorax if dull to percussion and prepare for chest drain insertion
        - six immediately life-threatening chest injuries (TOMCAT)
            - **t**ension pneumothorax
            - **o**pen pneumothorax
            - **m**assive haemothorax
            - **c**ardiac tamponade
            - **a**irway obstruction

                - **t**racheobronchial injury
        - circulation
            - look for any obvious bleeding and apply pressure
            - check heart rate
            - consider blood on the floor and four more (chest, abdomen, pelvis, long bones)
                - palpate abdomen for guarding and look for bruising
                - pelvic splint if concerns about pelvis injury
                - splint deformed femur
            - IV access
                - biggest cannula you can get in the antecubital fossa
                - bloods
                    - FBC, U&E, LFT, amylase, pregnancy test, G&S, clotting, VBG
                - IV fluids or blood as indicated
        - disability
            - AVPU or GCS assessment
            - any obvious head injuries
            - pain score and analgesia
        - exposure
            - remember to keep the patient warm
                - getting cold will worsen clotting
            - also consider dignity
            - secondary survey and identification of further injuries
                - expose a bit at a time and recover afterwards
                - likely to be done later after initial imaging
- silver trauma
    - older age generally considered 65 or over
        - International Consortium on Health Outcome Measures defines older age as the last 10 years of life before regional life expectancy
    - physiology may not change in the same way as in younger patients so they may not trigger a trauma pre-alert
        - e.g. baseline hypertension, beta-blockers
    - commonest mechanism for ISS >15 is a fall from standing height

RP5 respiratory failure

- background
    - occurs when disease of the heart or lungs leads to failure to maintain adequate blood oxygen levels (hypoxia) or increased blood carbon dioxide (hypercapnia)
        - hypoxaemic respiratory failure is characterised by a PaO_2 <8kPa with normal or low $PaCO_2$
        - hypercapnic respiratory failure is $PaCO_2$ >6kPa and PaO_2 <8kPa
    - may be acute, acute on chronic or chronic
- aetiology
    - common causes of type 1 respiratory failure
        - COPD
        - pneumonia
        - pulmonary oedema

        - pulmonary fibrosis
        - asthma
        - pneumothorax
        - pulmonary embolism
        - pulmonary hypertension
        - cyanotic congenital heart disease
        - bronchiectasis
        - acute respiratory distress syndrome
        - respiratory illness associated with HIV
        - kyphoscoliosis
        - obesity
    - common causes of type 2 respiratory failure
        - COPD
        - severe asthma
        - drug overdose/poisoning
        - myasthenia gravis
        - polyneuropathy
        - poliomyelitis
        - muscle disorders
        - head injuries and neck injuries
        - obesity
        - pulmonary oedema
        - adult respiratory distress syndrome
        - hypothyroidism
- presentation
    - symptoms
        - paroxysmal nocturnal dyspnoea and orthopnoea
        - confusion/reduced consciousness
        - shortness of breath
        - cough
    - signs
        - localised pulmonary findings determined by underlying cause
        - neurological features:
            - restlessness
            - anxiety
            - confusion
            - seizures
            - coma
        - tachycardia/cardiac arrhythmias
            - caused by hypoxaemia/acidosis
        - cyanosis
        - polycythaemia
            - present in longstanding hypoxaemia
        - cor pulmonale
            - right ventricular failure with hepatomegaly and peripheral oedema
- investigations
    - depend on cause and severity of respiratory failure and may include:

        - ABG
            - confirmation of diagnosis
        - CXR
            - often identifies the cause
        - FBC
            - anaemia can contribute to tissue hypoxia
            - polycythaemia may indicate chronic hypoxaemic respiratory failure
        - U&E and LFTs
            - may provide clues to the aetiology or indicate complications
            - electrolyte abnormalities (e.g. potassium, magnesium, phosphate) may exacerbate respiratory failure and other organ dysfunction
        - CK/troponin
            - to exclude recent MI
            - elevated CK may also indicate myositis
        - TFTs
            - hypothyroidism may cause chronic hypercapnic respiratory failure
        - spirometry/pulmonary function tests
            - useful in the evaluation of chronic respiratory failure
        - echocardiography
            - if cardiac cause suspected
        - ECG
            - cardiovascular cause
            - dysrhythmias from severe hypoxaemia or acidosis
        - microbiology
- management
    - hypoxaemia
        - aim for sats >90% with supplementary oxygen
        - beware prolonged high concentration oxygen in patients with chronic retention who rely on their hypoxic drive
        - assisted ventilation
            - mechanical ventilation
                - aims to support adequate gas exchange without harming the lungs
                - used to increase PaO_2 and decrease $PaCO_2$
                - rests the respiratory muscles where there is fatigue
                - weaning patients with chronic respiratory failure off mechanical ventilation can be difficult
            - non invasive ventilation
                - improves survival and reduces complications for selected patients with acute respiratory failure
                - main indications are:
                    - exacerbation of COPD
                    - cardiogenic pulmonary oedema
                    - pulmonary infiltrates in immunocompromised patients
                - can be used for weaning patients off invasive ventilation
            - extracorporeal membrane oxygenation (ECMO)

        - mainstay of therapy in neonatal and paediatric patients with life-threatening respiratory and/or cardiac failure
        - has also been used for adults with severe respiratory failure
    - strategies to support oxygenation can cause harm through:
        - lung stretch injury
        - oxygen toxicity
        - transfusion risks
        - cardiac over-stimulation
  - hypercapnia and respiratory acidosis
    - correct underlying cause and/or provide assisted ventilation
- complications
  - pulmonary
    - PE
    - pulmonary fibrosis
    - complications secondary to mechanical ventilation
  - cardiovascular
    - cor pulmonale
    - hypotension
    - reduced cardiac output
    - arrhythmias
    - pericarditis
    - acute myocardial infarction
  - gastrointestinal
    - haemorrhage
    - gastric distension
    - ileus
    - diarrhoea
    - pneumoperitoneum
    - duodenal ulceration caused by stress
  - polycythaemia
  - hospital acquired infection
    - pneumonia
    - urinary tract infection
    - catheter associated sepsis
  - renal
    - acute kidney injury
    - electrolyte and acid-base abnormalities
  - nutritional
    - malnutrition
    - complications related to enteral or parenteral nutrition or NG tube placement
- NIV
  - background
    - delivers oxygen to the lungs using pressure to reduce the amount of work the patient has to do to breathe
    - useful in both type 1 and type 2 respiratory failure
  - indications include:

        - pH <7.35
        - pCO_2 >6.5
        - RR >23
        - failure of medical therapy (e.g. steroids, nebulisers, controlled O_2)
    - absolute contraindications:
        - facial burns
        - fixed upper airway obstruction
        - facial deformity (mask will not fit)
        - pneumothorax
        - respiratory arrest
- mechanism
    - CPAP and BiPAP
        - CPAP (continuous positive airways pressure)
            - usually used by patients who use NIV at home as more portable
        - BiPAP (bilevel positive airways pressure)
            - tends to be used in hospital settings
            - can set a breathing rate to encourage the patient
            - can set the machine for IPAP and EPAP – more comfortable than CPAP where they have to exhale against a high pressure system
            - standard starting pressures:
                - IPAP 8-10mmHg
                - EPAP 2-4mmHg
            - blood gas should be repeated after an hour, an hour after any change in settings and after 3 and 12 hours of treatment
            - if pO_2 still low, turn up oxygen or turn up EPAP 2-3mmHg at a time to max 12mmHg
                - consider IPAP up to max 20mmHg if estimated volumes less than estimated for patient's size
            - if pO2 high, turn down oxygen, then decrease EPAP
            - if CO2 high, consider increasing ventilatory rate then turn up IPAP to increase ventilatory volume delivered
                - rough aim is 5-7ml/kg ideal body weight
        - when the patient exhales, the alveoli do not completely collapse because some form of pressure is continuously delivered
            - more energy efficient than trying to open up closed airways and means oxygen delivery starts earlier because the airway is already open during inhalation
        - nebulised drugs can be given through the circuit
        - if the patient is agitated, reassurance can be attempted and light sedatives given (e.g. 0.25mg lorazepam)
    - summary for setting up NIV
        - PPE
        - confirm indication
            - is it appropriate?
            - is there any pneumothorax on CXR or US?
            - ceiling of care set
            - medically optimised

        - prepare and inform patient
            - explain procedure to patient and that it is to help get oxygen into the lungs
            - keep dentures in to improve mask fit
            - if anticipating use over >24 hours, put pressure relieving dressing over bridge of nose
        - prepare NIV
            - size mask
            - connect tubing
            - connect oxygen tubing
            - confirm settings (IPAP 8-10mmHg, EPAP 2-4mmHg)
        - aerosol generating PPE with FFP3 mask and visor
        - turn on NIV machine
            - mute disconnect alarm
            - hold mask to patient's face
            - use assistant if possible to attach mask to face
            - reassurance
            - run hand around mask to feel for leaks
            - check settings
            - reassurance
        - leave patient area and remove PPE
        - complete paperwork
            - NIV prescription
            - documentation
        - aerosol generating PPE
        - review
            - review patient
            - repeat ABG 1 hour after starting
            - offer patient a drink
        - adjust NIV settings
            - if pO_2 still low – turn up O_2 or turn up EPAP up to max 12; consider increasing IPAP
            - if pO_2 high, turn down oxygen then turn down EPAP
            - if CO_2 high, increase ventilatory rate; turn up IPAP

RP6 sepsis

- background
    - sepsis is a life-threatening organ dysfunction due to a dysregulated host response to infection
    - septic shock is associated with profound circulatory, cellular and metabolic abnormalities and have a vasopressor requirement to maintain MAP 65mmHg or above in the absence of hypovolaemia
- pathophysiology
    - derangement in physiology may include:
        - abnormal coagulation
        - endothelial cell dysfunction
        - presence of excessive tumour necrosis factor

        - cell apoptosis (e.g. lymphocytes and endothelial cells)
        - neutrophil hyperactivity
        - poor glycaemic control
        - lack of steroid hormones
- risk factors
    - there is usually an abscess or nidus of infection, which may be occult
    - risk factors for developing sepsis include:
        - age (over 75 years or under 1 year)
        - instrumentation or surgery
        - indwelling line or catheter
        - alcohol misuse
        - diabetes mellitus
        - breach of skin integrity (e.g. burns)
        - immunocompromise
        - medications (e.g. high dose steroids, chemotherapy)
        - males are more prone to develop severe sepsis but the mortality in females is higher
        - IV drug misuse
        - pregnancy
- presentation
    - features may be non-specific with a high degree of suspicion required
    - features to consider include:
        - patients presenting a few days earlier with a focus of infection and a rapid deterioration despite appropriate oral antibiotics
        - non-specific symptoms
            - e.g. lethargy, nausea and vomiting, abdominal pain, diarrhoea
        - symptoms relating to a possible focus of infection
            - e.g. cough, urinary symptoms, recent travel
        - frequency of micturition in past 18 hours
        - presenting features in children may include:
            - feeling abnormally cold to the touch
            - looking mottled or blue
            - very pale skin
            - non-blanching rash
            - raised respiratory rate
            - lethargy
            - difficult to wake up
        - young children may not feed, may have repeated vomiting and may not pass urine so have no wet nappies
    - the qSOFA score is not used for diagnosis of sepsis but as a mortality predictor
        - altered mental status (GCS <15)
        - respiratory rate ≥22
        - systolic BP ≤100
        - (a score of >1 is high risk for in-hospital mortality and a full SOFA score should be considered – generally used on ITU)
- investigations
    - FBC

        - may show anaemia, neutrophilia, neutropenia, thrombocytopenia
        - lymphocytosis in viral infections
    - urine
        - dipstick +/- microscopy, culture and sensitivities
        - consider if symptomatic or if no other source identified
    - renal function
        - extent of dehydration and organ failure
    - LFTs
        - likely to show hypoalbuminaemia
    - glucose
        - may have hyperglycaemia
    - clotting
        - including d-dimer and fibrinogen to assess for DIC
    - blood cultures
        - at least two
        - consider cultures for mycobacteria
        - ideally should be sent before antibiotics are given
    - radiology
        - CXR
        - abdominal ultrasound looking for a collection
        - CT scan looking for a source
    - VBG or ABG
    - more invasive investigations may be required, e.g.:
        - lumbar puncture
        - bronchoscopy
        - laparoscopy
        - lymph node biopsy
- management
    - sepsis six should be delivered within the first hour
        - high-flow oxygen
        - blood cultures
        - IV antibiotics
        - IV fluid resuscitation
        - haemoglobin and serial lactates
        - hourly urine output measurement
    - general principles of hospital care (NICE Quality Standards)
        - patients fulfilling one or more of the criteria for high risk of severe illness or death should receive the first dose of IV antibiotics and senior clinician review immediately, and at least within an hour
        - those at risk who require treatment for cardiovascular instability should receive a fluid bolus within one hour of risk stratification
        - patients receiving IV antibiotics or a fluid bolus who do not respond within one hour of treatment should be seen by a consultant
        - patients assessed as low risk for severe illness or death should be educated about symptoms to monitor and accessing medical care
    - specific therapy
        - International Guidelines for Management of Severe Sepsis and Septic Shock (2012):

- IV antibiotics
    - broad spectrum
    - antivirals and antifungals may also be required (e.g. in immunocompromised patients)
    - empirical combination therapy should not be administered for more than 3-5 days with de-escalation to the most appropriate single therapy once sensitivities known
- IV fluid resuscitation
    - crystalloid
    - consideration of the addition of albumin in patients requiring substantial amounts to maintain MAP
    - initial fluid challenge for tissue hypoperfusion and hypovolaemia should be a minimum of 30ml/kg, continued as long as there is haemodynamic improvement
- vasopressor support
    - noradrenaline infusion as first choice to maintain MAP >65mmHg
    - adrenaline when an additional agent is required
    - vasopressin can be added to raise MAP or decrease noradrenaline dose
    - dobutamine infusion in the presence of myocardial dysfunction or ongoing signs of hypoperfusion despite achieving adequate intravascular volume and adequate MAP
    - avoid IV hydrocortisone if adequate fluid resuscitation and vasopressor therapy are able to restore haemodynamic stability
- PEEP
    - for acute respiratory distress syndrome
- blood glucose management
    - with insulin
- other management
    - prophylaxis for DVT
    - stress ulcer prophylaxis
    - oral or enteral feeding
- surgery if needed
    - e.g. wound debridement, abscess drainage

- prognosis
    - mortality rate of severe sepsis in the UK is 35% (over 40% in severe septic shock)
    - each hour of delay in antibiotics over the next 6 hours is associated with a decrease in survival of 7.6% for patients with septic shock
    - lactate level is associated with in-hospital mortality
    - prognosis is worse in the elderly
    - factors significantly associated with long term mortality:
        - congestive heart failure
        - peripheral arterial disease
        - dementia

        - diabetes with complications
        - use of mechanical ventilation
- complications
    - DIC
    - adrenal failure
        - e.g. adrenal failure secondary to meningococcus (Waterhouse-Friderichsen syndrome)
    - multi-organ failure
    - post-sepsis syndrome
        - physical and psychological difficulties that may last for years
        - physical problems may include:
            - lethargy
            - muscle weakness
            - breathlessness
            - chest pains
            - oedema
            - arthralgia
            - poor appetite
            - visual disturbance
            - sensory disturbance
            - recurrent infections
        - psychological problems
            - anxiety
            - depression
            - PTSD
            - nightmares
            - insomnia
            - poor concentration
            - memory disturbance
- RCEM sepsis standards
    - observations
        - on arrival:
            - temperature
            - heart rate
            - respiratory rate
            - blood pressure
            - mental status
            - blood glucose
    - review
        - senior EM review within an hour
    - treatment
        - high flow O2 before leaving ED
        - 20ml/kg fluid
            - 75% within an hour of arrival
            - 100% before leaving ED
        - antibiotics
            - 50% within an hour

                - 100% before leaving ED
        - investigations
            - before leaving ED
                - lactate
                - blood cultures
                - measurement of urine output

RP7 shock

- definition
    - a state that results when circulatory insufficiency leads to inadequate tissue perfusion and thus delivery of oxygen to the tissues of the body
- categories
    - hypovolaemic
        - inadequate circulating volume secondary to fluid loss
        - causes include:
            - haemorrhagic, e.g.:
                - trauma
                - GI bleed
                - obstetric haemorrhage
                - ruptured AAA
            - diarrhoea and vomiting
            - diabetic ketoacidosis
            - burns
    - distributive
        - inadequate perfusion secondary to maldistribution
        - causes include:
            - sepsis
            - neurogenic shock
            - anaphylaxis
    - obstructive
        - inadequate cardiac output as a result of mechanical obstruction
        - causes include:
            - pulmonary embolism
            - tension pneumothorax
            - cardiac tamponade
            - acute IVC or SVC obstruction
    - cardiogenic
        - inadequate cardiac output as a result of cardiac failure
        - causes include:
            - myocardial infarction
            - myocardial contusion
            - myocarditis
            - late sepsis
            - overdose (e.g. beta-blockers)
            - complete heart block
- pathophysiology
    - oxygen delivery

        - global oxygen delivery is the amount of oxygen leaving the left ventricle every minute
        - determined by cardiac output (CO) and arterial oxygen content (CaO_2)
            - CO is heart rate (HR) x stroke volume (SV)
        - the vast majority of oxygen in the blood is bound to haemoglobin
            - only a tiny proportion (measured by PaO_2) is in solution
        - the main determinants of arterial oxygen content are oxygen saturation and haemoglobin concentration
        - global oxygen delivery can be calculated as:
            - (HR x SV) x [Hb]g/dl x 10 x 1.34 x sO_2ml/L
                - the 10 converts Hb to g/L
                - the 1.34 is the amount of oxygen in ml carried by 1 gram of 100% saturated haemoglobin
    - inadequate delivery
        - cells run on adenosine triphosphate (ATP)
        - ATP is produced by respiration (either aerobic or anaerobic)
            - anaerobic respiration is around 18 times less efficient
        - in the absence of adequate oxygen delivery, cells run out of energy and cease to function effectively, and die if the situation is not corrected
            - shows on the macroscopic level as organ dysfunction and failure
        - the body can compensate by dramatically increasing the amount of oxygen extracted from the blood (oxygen extraction ratio)
            - initial clinical signs and symptoms of shock are the result of the compensatory mechanisms
            - the later features are those of organ dysfunction as the compensatory mechanisms fail
                - this will become irreversible if not treated
- assessment
    - ABCDE approach
    - four concurrent steps:
        - recognition of the degree of physiological compromise
        - identification of the cause
        - correction of the physiological deficit
        - treatment of the underlying cause
    - heart rate
        - reduction in cardiac output causes reduced activity of arterial baroreceptors causing increased sympathetic and decreased parasympathetic activity
            - heart rate increases and cardiac output is restored towards normal
        - heart rate does not always increase
            - absence of tachycardia does not exclude significant compromise
            - around a third of patients with significant blood loss present with an initial relative bradycardia (<100 bpm)
            - patients on beta-blockers cannot mount a tachycardia
            - athletes may have an unrecognised tachycardia as their normal heart rate is so slow
            - bradycardia may also be the cause of the shock state (e.g. complete heart block, calcium-channel blocker overdose)

        - neurogenic shock usually has an absence of tachycardia
    - skin
        - reduced CO vasoconstriction caused by increased sympathetic activity diverts blood away from the peripheral circulation leading to cool skin
        - capillary refill is useful in children but of questionable value in adults
        - there may be sweating as a result of increased sympathetic activity leading to clamminess
        - in distributive shock the skin may be warm and dry
        - neurogenic shock (loss of sympathetic tone as a result of cord injury) leads to vasodilation and an absence of sweating
            - occurs in around 20% of patients with cervical cord injury at presentation
        - in anaphylaxis there may be patchy or generalised erythema, urticaria and angioedema
            - skin or mucosal changes may be absent in up to 20%
        - in early sepsis the skin may be warm but as the condition worsens peripheral perfusion is reduced
            - there may be purpura in meningococcal sepsis and generalised erythema in toxic shock syndrome
    - respiratory rate
        - excellent marker of physiological compromise
        - may be increased because of hypoxia
        - often remains elevated despite correction of CaO_2 as worsening perfusion generates a respiratory acidosis requiring respiratory compensation
    - blood pressure
        - maintained until late stages of shock due to compensation
        - MAP is a better indicator of organ perfusion than the systolic
            - MAP = (systolic + (2 x diastolic))/3
            - a map of 65mmHg is considered sufficient for organ perfusion in a healthy adult
    - conscious level
        - may be reduced for various reasons in unwell patients
        - may be a result of inadequate cerebral perfusion
    - urine output
        - little use in initial assessment
        - any patient who is shocked should be catheterised early to provide an indication of the adequacy of resuscitation over time
        - urine output may be misleading in some cases, such as an osmotic diuresis in DKA
- investigations
    - focused on identifying the cause of the shock
        - e.g. FAST scan in trauma
        - ECG in cardiogenic shock
        - echo in massive PE
    - blood lactate
        - generated by anaerobic respiration caused by falling oxygen delivery

        - lactate >4 is associated with increased ICU admission and mortality in normotensive patients with sepsis
        - those with a higher lactate clearance at 6 hours have a better outcome
        - base excess and bicarbonate levels offer some guidance as to the level of compromise and degree of resuscitation but can remain normal even with significantly abnormal lactate levels
    - central venous oxygen saturation
        - oxygen saturation of blood falls as oxygen is extracted so the saturation of blood returning to the lungs (from the pulmonary artery) (SvO_2) can give an indication of total body extraction
            - mixed venous blood is usually around 70-75% saturated
                - if it falls below this, oxygen extraction has had to increase
                - in shock states it is usually because oxygen delivery has become inadequate
                - sampling in the ED is not practical
        - central venous oxygen saturation ($ScvO_2$) is used as a surrogate
            - tends to be about 5-7% higher
            - its use is supported by the Surviving Sepsis Campaign
            - can be misleading, particularly in sepsis
                - oxygen extraction by the tissues becomes less and less efficient and blood returning to the heart remains oxygenated
                - a normal or high $ScvO_2$ in this situation can reflect a worsening clinical picture
- management
    - treatment of the underlying condition
    - reversal of the physiological deficit (resuscitation)
    - frequent reassessment after each intervention
        - response will be rapid, transient or none
    - if response is inadequate consider:
        - adequacy of resuscitation so far
        - accuracy of diagnosis
        - need for immediate definitive treatment (e.g. decompression of tension pneumothorax)
        - need for more invasive cardiac monitoring
    - early goal directed therapy
        - an early aggressive approach is usually indicated
        - caution in bleeding patients where haemostasis has not yet been achieved
        - may be counterproductive if the shock state is recognised too late
        - in the majority of cases, it will consist of oxygen and fluid boluses
            - small volumes (250ml) given quickly (5-10 minutes) with reassessment after each
        - blood transfusion to a target of 7-9g/dl in otherwise healthy non-trauma patients to optimise oxygen delivery
    - inotropes
        - have a role in some presentations (e.g. sepsis, cardiogenic shock, neurogenic shock, anaphylaxis) but only once adequately resuscitated for hypovolaemia

- intubation and ventilation
  - should be considered early
  - oxygen consumption can be dramatically reduced by taking over the work of breathing
  - in sepsis, increased capillary permeability increases the risk of pulmonary oedema with necessary fluid resuscitation
    - in the paediatric population, it is recommended that intubation is considered once fluid resuscitation exceeds 40-60ml/kg
- steroids
  - only in adrenal crisis (which should be considered in any unexplained hypotension)

RP8 unconsciousness

- background
  - there is no single examination finding, score or test that distinguishes between all patients in a coma and all who are not
  - level of unresponsiveness depends on how much of the brain is functioning and the intensity of the stimulus
- pathophysiology
  - consciousness requires two key components of the CNS to be functioning: the reticular activating system (RAS) and at least one cerebral hemisphere
    - for a person to be unconscious, either the RAS or both cerebral hemispheres have ceased functioning
  - main causes of failure of the RAS:
    - brain stem stroke (ischaemic or haemorrhagic)
    - pre-death event: oedema pushing on the brainstem and causing it to fail
  - failure of both cerebral hemispheres:
    - failure of an adequate blood supply
    - inadequate substrate for normal metabolism (e.g. oxygen, glucose)
    - direct or indirect trauma to the cerebrum
    - exposure of the brain to a toxic insult including:
      - infection
      - toxic metabolites
      - exogenous poisons
- assessment
  - check for and correct hypoxia and hypovolaemia
  - evidence of injury
    - scalp laceration
    - scalp haematoma
    - signs of basal skull fracture
    - bitten tongue suggesting seizure
  - evidence of fever
    - bacterial meningitis
    - encephalitis
    - brain injury (pontine or hypothalamic)
    - hypothermia may indicate:
      - sepsis

- exposure to cold
- intoxication
- hypothyroidism

- evidence of organ failure
  - hypoxia and hypercapnia associated with hypoventilation
  - hypotension associated with cardiac failure
  - examination of breath for:
    - ketones (pancreatic failure)
    - uraemia (renal failure)
    - fetor hepaticus (liver failure)
    - garlic (organophosphate/insecticide poisoning)
- evidence of toxic ingestion/inhalation
  - needle marks associated with IV drug abuse
  - bullae associated with barbiturate overdose
  - cherry red appearance of carbon monoxide poisoning
  - dry skin (especially in normally moist areas) suggesting tricyclic overdose/other anticholinergics
  - profuse sweating indicates hypoglycaemia or organophosphate poisoning
- detailed neurological assessment
  - GCS to determine depth of coma
    - GCS scale
      - infant <1 year
        - eyes
          - 4: open
          - 3: to voice
          - 2: to pain
          - 1: no response
        - verbal
          - 5: coos, babbles
          - 4: irritable cry, consolable
          - 3: cries persistently to pain
          - 2: moans to pain
          - 1: no response
        - motor
          - 6: normal spontaneous movement
          - 5: withdraws to touch
          - 4: withdraws to pain
          - 3: decorticate flexion
          - 2: decerebrate extension
          - 1: no response
      - child 1-4 years
        - eyes
          - 4: open
          - 3: to voice
          - 2: to pain
          - 1: no response
        - verbal

                - 5: oriented, speaks, interacts, social
                - 4: confused speech, disoriented, consolable
                - 3: inappropriate words, inconsolable
                - 2: incomprehensible, agitated
                - 1: no response
            - motor
                - 6: normal spontaneous movement
                - 5: localises pain
                - 4: withdraws to pain
                - 3: decorticate flexion
                - 2: decerebrate extension
                - 1: no response
        - 4 years – adult
            - eyes
                - 4: open
                - 3: to voice
                - 2: to pain
                - 1: no response
            - verbal
                - 5: oriented and alert
                - 4: disoriented
                - 3: nonsensical speech
                - 2: moans, unintelligible
                - 1: no response
            - motor
                - 6: follows commands
                - 5: localises pain
                - 4: withdraws to pain
                - 3: decorticate flexion
                - 2: decerebrate extension
                - 1: no response
    - patients with GCS <10 who are not likely to recover quickly should be assessed for the need for intubation to protect the airway and optimise respiratory function
    - record pupillary responses to light, ocular abnormalities and limb posturing
    - where head injury is not suspected, assess for neck stiffness
- differential
    - evidence of head injury
        - priority is to maintain cerebral perfusion pressures and cerebral oxygenation
        - hypotension and hypoxia should be aggressively treated
        - urgent CT scan required to look for a neurosurgically remediable cause
    - no head injury but focal neurological signs
        - likely acute cerebrovascular event
            - most patients with an acute cerebrovascular event do not present with coma and other causes should be sought
        - must rule out hypoglycaemia
    - no head injury, no neurological signs, infection probable

        - differential is meningitis or cerebral malaria
        - priority is to start antibiotics and undertake malaria testing if it is a possible differential
        - urgent CNS imaging is required
    - no head injury, no neurological signs, infection unlikely
        - can be regarded as having been poisoned
        - may be exogenous (e.g. carbon monoxide, tricyclic antidepressants, narcotics) or endogenous as a result of end organ failure (e.g. DKA, myxoedema coma, respiratory failure)
        - most useful tests are ABG, serum anion gap and serum electrolytes
    - intracranial causes include:
        - intracerebral abscess
            - planned surgical drainage required
        - cerebellar haemorrhage
            - patients with cerebellar haemorrhage >3cm have better outcomes if the haematoma is surgically drained
            - intervention before the onset of hydrocephalus and/or loss of brainstem reflexes improves outcome
        - metastasis/primary tumour
            - may or may not be amenable to surgery
        - intracerebral bleed
            - there is no evidence that surgical evacuation improves outcome
- management
    - oxygen for all patients
    - manage hypotension
    - consider reversal agents (e.g. naloxone, flumazenil)
        - usually better to support the airway and ventilation until the agents wear off rather than risk withdrawal/seizures
    - thiamine for alcoholics/starved patients – Wernicke's can cause acute exacerbation of neurological damage
- prognosis
    - 3 main CNS causes of death are:
        - raised intracranial pressure
        - herniation
        - encephalitis/meningitis
    - prognosis is determined primarily by the cause of the coma
    - coma following cardiac arrest is not of itself an indication to withdraw therapy
- pitfalls
    - considerations:
        - locked in syndrome
            - an acute pontine lesion causes paralysis of all 4 limbs and the head and neck including the facial muscles
            - the patient is conscious and is able to blink and move their eyes because the oculomotor pathways are spared
            - ask the patient to blink and move their eyes up and down before applying a painful stimulus
        - malingering

- diagnosis of exclusion
- patients may or may not be convincing
- often the history raises suspicion and neurological findings may be contradictory or inconsistent
- normally lasts a day or so with a characteristic sudden awakening and associated retrograde amnesia

RC1 choking

- how to manage (adult resus council)
    - mild airway obstruction (effective cough)
        - encourage cough
        - continue to check for deterioration to ineffective cough or until obstruction relieved
    - severe airway obstruction (ineffective cough)
        - conscious
            - 5 back blows
            - 5 abdominal thrusts
        - unconscious
            - start CPR
- how to manage (child resus council)
    - effective cough (crying or verbal response to questions, loud cough, able to take a breath before coughing, fully responsive)
        - encourage cough
        - continue to check for deterioration to ineffective cough or until obstruction relieved
    - ineffective cough (unable to vocalise, quiet or silent cough, unable to breath, cyanosis, decreasing level of consciousness)
        - conscious
            - 5 back blows
            - 5 thrusts
                - chest for infant
                - abdominal for child >1 year
        - unconscious
            - open airway
            - 5 breaths
            - start CPR

RC2 stridor

- definition
    - loud, harsh, high pitched respiratory sound
        - may start as low pitched croaking and progress to high pitched crowing on more vigorous respiration
    - usually heard on inspiration due to partial obstruction of airway
        - usually extrathoracic (trachea, larynx, pharynx)
    - can occur on expiration in severe upper airway obstruction
        - usually indicated tracheal or bronchial obstruction (intrathoracic)
    - biphasic stridor suggests subglottic or glottic obstruction

- epidemiology
    - common in younger children with smaller airways
        - often accompanies upper respiratory tract infection
        - chronic stridor usually occurs with congenital conditions
    - much less common in adults
        - chronic stridor in adults often indicates serious underlying pathology
- causes in children
    - croup/laryngotracheobronchitis
        - most common cause
        - usually between 6 months and 2 years
        - barking, seal-like cough, low fever, worse at night
    - inhaled foreign body
        - common, especially in children age 1-2 years
        - preceded by choking or coughing
    - tracheitis
        - uncommon
        - usually under 3 years
        - bacterial infection following a viral infection in toddlers
    - abscesses
        - may be retropharyngeal (under 6 years)
        - may be peritonsillar (usually in adolescents)
        - presents with high fever and difficulty in swallowing
        - retropharyngeal abscesses present with pain on swallowing and hyperextension of the neck
        - peritonsillar abscess presents with trismus, difficulty with swallowing and difficulty with speaking
    - anaphylaxis
        - hoarseness and inspiratory stridor
        - accompanied by symptoms of an allergic reaction
        - usually within 30 minutes of exposure to an allergen
    - epiglottitis
    - congenital problems
        - laryngomalacia
            - most common cause of stridor
            - occurs in neonates and early infancy
            - often exacerbated in supine position, by crying and by feeding
        - vocal cord dysfunction
            - next most common cause in infants
            - biphasic and associated with a weak cry
            - unilateral vocal cord palsy is most common and can be secondary to birth trauma or intrathoracic surgery
            - usually resolves in first 2 years of life
        - subglottic stenosis
            - may be congenital with narrowing of the subglottis and cricoid rings
            - can be acquired after prolonged intubation
            - causes inspiratory stridor but can be biphasic and misdiagnosed as asthma

        - laryngeal disorders
            - congenital laryngeal webs can cause biphasic stridor
            - other disorders include laryngeal dyskinesia and exercise-induced laryngomalacia
            - laryngeal tumours such as cysts, haemangiomas or papillomas
        - tracheomalacia
            - caused by external compression or defective tracheal cartilage
            - most common cause of expiratory stridor
        - choanal atresia
            - most common congenital abnormality of the nose in infants
            - unilateral may be symptomatic
            - bilateral may present with apnoea or cyanosis during feeding
            - can be diagnosed by an inability to pass a nasal catheter
        - tracheal stenosis
            - usually caused by tracheal rings and presents with persistent stridor and a prolonged expiratory phase
            - other causes include external compression from aortic arch abnormalities
- causes in adults
    - airway trauma
        - can present with stridor and sudden onset of dysphonia and haemoptysis
        - signs include cyanosis, intercostal recession, nasal flaring, tachypnoea and progressive dyspnoea with shallow respirations
        - can be surgical emphysema in neck or upper chest
    - anaphylaxis
        - causes stridor with upper airway oedema and laryngospasm
    - acute laryngitis
        - stridor caused by severe laryngeal oedema
        - usually accompanied by hoarseness
    - aspiration of foreign body
        - life threatening sudden onset of stridor
        - may also be paroxysmal coughing, gagging or choking, hoarseness, wheezing, tachycardia and other signs of respiratory distress
        - patients are usually anxious and distressed
    - acute epiglottitis
        - rare in adults
    - retropharyngeal abscess
    - laryngospasm
        - in hypocalcaemia, accompanied by paraesthesia and other signs of calcium deficiency
        - inhalation injury
            - singed nasal hairs, burns around the face, cough, hoarseness, sooty sputum, crackles, rhonchi, wheeze, signs of respiratory distress
- chronic stridor in adults
    - laryngeal tumour
        - stridor is a late sign
    - laryngeal inflammation

        - diphtheria
        - tuberculosis
        - syphilis
        - sarcoidosis
        - granulomatosis with polyangiitis
    - cricoarytenoid ankylosis
        - in rheumatoid arthritis
    - tumours causing compression
        - mediastinal tumours
        - retrosternal thyroid
            - with tracheal deviation and signs of thyrotoxicosis
        - thoracic aortic aneurysm
    - iatrogenic causes
        - bronchoscopy/laryngoscopy
        - prolonged intubation
        - neck surgery
- history
    - children
        - age of onset
        - duration, progression and severity of stridor
        - precipitating factors (feeding, crying)
        - whether positional (which position worse)
        - whether aphonia present
        - other symptoms (cough, aspiration, drooling, choking, cyanosis, sleep)
        - severity (colour change, respiratory effort, apnoea)
        - perinatal history
        - developmental history
        - vaccination history
        - growth and weight gain
    - adults
        - onset, duration, progression and severity
        - past medical history and details of any trauma or surgery
- examination
    - observation
        - fever and signs of toxicity indicating bacterial infection
        - drooling
        - character of cry, cough and voice
        - in children, craniofacial features, nasal patency and cutaneous haemangiomas
        - any positional preference that alleviates stridor
    - palpate (carefully)
        - crepitations or masses in neck, face or chest
        - deviation of trachea
    - auscultate
        - nose, oropharynx and chest (can help locate source of stridor)
- investigations
    - may not be required in self limiting upper respiratory tract infection
    - pulse oximetry

    - ABG
    - imaging
        - AP and lateral x-rays of neck and chest
        - special view x-rays (inspiratory/expiratory and lateral decubitus to demonstrate air trapping)
        - contrast studies
            - if compression, trachea-oesophageal fistula or gastro-oesophageal reflux suspected
        - CT
            - for aberrant vessels and mediastinal masses
        - MRI
            - particularly for upper airway and vascular abnormalities
        - virtual bronchoscopy
        - laryngoscopy and bronchoscopy once oxygen sats stable and acute epiglottitis excluded
- management
    - depends on cause
    - may require urgent airway management
    - medications from corticosteroids to antibiotics may be useful
    - surgical procedures may be required

RC3 organ donation

- types of donation
    - deceased organ donation
        - can include kidneys, heart, liver, lungs, pancreas, small bowel, corneas, other tissues (heart valves, skin, bone, tendons)
        - donation after brainstem death
            - death diagnosed by brainstem tests whilst patient on ventilator
                - irreversible condition
                - complicating medical conditions excluded
                    - severe electrolyte abnormality
                    - acid-base derangement
                    - endocrine disturbance
                    - haemodynamic compromise
                    - drug intoxication/poisoning
                    - hypothermia
                    - locked-in syndrome
                    - Guillain-Barré syndrome
                - two separate examinations performed by different physicians at least an hour apart
                - brain stem reflexes
                    - pupils (CN II sensory, III constriction)
                        - no response to light, fixed and mid-dilated
                    - ocular movement (CN VIII sensory, VI lateral rectus)
                        - oculocephalic reflex – positive doll's eyes
                            - eyes should move to maintain forward fixation as head turned

- oculovestibular reflex
    - irrigation with cold water onto tympanic membrane, observe for deviation of eyes towards cold ear if reflex not intact/fast beating nystagmus to the opposite side if reflex intact)
- corneal reflex (CN V sensory, VII motor/blinking)
    - saline flush dripped into eye
    - direct stimulation with gauze if no response to saline
- pharyngeal (CN IX/X)
    - gag – tongue blade or suction
- tracheal (CN X)
    - endotracheal suction in intubated patients
- spontaneous body movements may represent spinal reflexes and should not be misinterpreted as brain stem function

- apnoea testing involving disconnecting from the ventilator and observing no respiratory effort despite significant rise in CO_2

- donation after circulatory death
    - potential donor who dies in hospital but is not on a ventilator
    - organs to be donated must be removed within a few minutes of the heart stopping to prevent hypoxic damage
- living organ donation
    - usually involves a family member donating an organ
        - may be an unrelated altruistic donor
    - kidneys are the most common organ donated
    - part of the liver, bone and amniotic membrane (used in eye surgeries to promote wound healing) can be donated
        - the placenta with the amniotic membrane can be donated when the baby is delivered by c-section

- identification of potential donors
    - patient receiving end of life care, has had a catastrophic brain injury, has absence of one or more cranial nerve reflexes and a GCS score of 4 or less not explained by sedation
    - it is expected that withdrawal of life-sustaining treatment would result in circulatory death
    - discussion with the specialist nurse for organ donation (SN:OD) should be started
- best interests
    - if patients lack capacity, a best interests decision should be made
    - life-sustaining treatments should be continued whilst this is explored
    - things to consider include:
        - what is known about the patient's views, including advance statements or registration on the NHS Organ Donation Register, or views expressed to friends and family

        - beliefs or values likely to influence the patient if they had capacity
        - any other factors likely to influence the patient if they had capacity
        - the views of family, friends and anyone else involved in the provision of care
        - anyone identified by the patient to be consulted
- approaching relatives
    - should not be done by the ED team regarding organ donation
    - requires an MDT (including the medical and nursing team caring for the patient, the SN:OD and any appropriate faith representatives)
    - before approaching the family, the MDT should look into:
        - potential for donation
        - NHS Organ Donor Register, Lasting Powers of Attorney for health and welfare, advance statements
        - coronial, legal and safeguarding issues
        - clinical history
        - identity of key family members
        - need for family support – e.g. faith/advocate/translator

RC4 BRUE

- definition
    - Brief Resolved Unexplained Event
        - diagnosis of exclusion
    - an episode in an infant <12 months:
        - duration <1 minute (typically 20-30 seconds)
        - sudden onset with a return to baseline state
        - characterised by ≥1 of:
            - cyanosis or pallor
            - absent, decreased or irregular breathing
            - marked change in tone (hypertonia or hypotonia)
            - altered level of consciousness
        - not explained by identifiable medical conditions
- differential
    - physiological
        - transient choking/gagging
        - apnoea of prematurity
    - cardiac
        - congenital heart disease
        - arrhythmia
        - prolonged QT
    - gastrointestinal
        - reflux
    - respiratory
        - airway obstruction including inhaled foreign body
        - laryngomalacia
        - obstructive sleep apnoea
        - central sleep apnoea
    - infection
        - whooping cough

        - URTI/LRTI
        - meningitis/encephalitis
        - UTI
        - gastroenteritis
    - CNS
        - head injury
        - epilepsy
        - structural cerebral abnormalities
        - neuromuscular disorders
    - NAI
        - drug ingestion
        - factitious illness
        - suffocation
        - inflicted injury
    - surgical
        - acute obstruction
    - metabolic/toxins
        - hypoglycaemia
        - hypocalcaemia
        - hypokalaemia
        - inborn errors of metabolism
        - intentional/non-intentional drug overdose
- history (from the person who witnessed the event)
    - description of event
        - choking, gagging
        - breathing: struggling to breathe, pauses, apnoea
        - colour and colour distribution: normal, cyanosis, pallor, plethora
        - distress
        - conscious state: responsive to voice, touch or visual stimulus
        - tone: stiff, floppy or normal
        - movement (including eyes): purposeful, repetitive
    - circumstances and environment prior to event
        - awake or asleep
        - relationship of the event to feeding and history of vomiting
            - amount and type of feed
            - history of reflux
        - position (prone, supine, side)
        - environment: sleeping environment, co-sleeping, temperature, bedding, smokers in the house
        - objects nearby that could be swallowed, cause choking, suffocation
        - illness in preceding days
        - preceding trauma or recent head injury
    - end of event
        - duration of event
        - circumstances of cessation: self-resolved, repositioned, stimulation, mouth to mouth, chest compressions
        - recovery phase: rapid or gradual
    - other history

        - birth history
            - gestational age (infants born prematurely and younger than 44 weeks corrected age can have apnoea of prematurity)
            - perinatal history, including SCBU/NICU
        - past medical history including previous similar events
        - preceding/intercurrent illness
        - sick contacts
        - family history of sudden death or significant childhood illness
            - parental consanguinity
        - patient medications, medications or other drugs within the home
        - social history: parental support, psychosocial assessment
- examination
    - full A-E assessment
        - consider differentials
        - check glucose
    - full exposure
        - bruising
        - bleeding from nose/mouth
        - torn frenulum
        - subconjunctival haemorrhage
    - plot weight, length and head circumference
    - note any dysmorphic features
- risk assessment
    - low risk if:
            - >60 days of age
            - born >32 weeks' gestation and have a corrected age of >45 weeks (or approximately >2 months)
                - corrected age = chronological age – weeks of prematurity
            - no CPR given by trained practitioner
            - <1 minute duration
            - first event
        - they are unlikely to have a serious underlying condition and it is unlikely to recur
    - high risk
        - any patient who does not fit the low risk group
- investigations
    - low risk group
        - none required (except glucose)
        - some units do ECG and capillary gas
    - high risk group
        - ECG
        - capillary gas
        - cardiorespiratory monitoring
- management
    - low risk patients can be discharged
        - they are sometimes admitted for a period of monitoring and parental education

- discharge advice should include:
    - safe sleeping
        - Lullaby Trust has printable information
        - e.g sleeping on back, individual cot without toys/bumpers/loose bedding in same room as parents, breastfeed if possible, room temperature 16-20 degrees
    - eliminating exposure to tobacco smoke
    - infant BLS
    - specific advice never to shake the infant in an attempt to resuscitate them
    - GP follow up in the next 48 hours
- high risk patients
    - admit to paediatrics for 24 hours for monitoring and investigations

RC5 SUDIC protocol

- background
    - all Local Safeguarding Children's Boards (LSCB) are required to review the deaths of all children in their area
    - the SUDIC process describes the process of communication, collaborative action and information sharing following the unexpected death of a child
    - an unexpected death is defined as:
        - 'the death of a child which was not anticipated as a significant possibility, for example 24 hours before the death, or where there was a similarly unexpected collapse or incident leading to or precipitating the events that led to the death'
    - all agencies need to follow five common principles:
        - sensitivity
        - open mind/balanced approach
        - interagency response
        - sharing of information
        - appropriate response to the circumstances and preservation of evidence
- action in the ED
    - unless the infant has clearly been dead for some time, with signs of rigor mortis, resuscitation should be attempted on arrival at the hospital
        - it should be continued according to APLS until a full assessment has been made, taking account of the clinical features, response to appropriate resuscitation and any immediately available results of investigations
    - when to stop resuscitation
        - no return of circulation at any time up to 30 minutes of cumulative life support
        - or until an experienced senior clinician has made the decision to stop
    - even when the infant has clearly been dead for some time it is often helpful for them to be brought to the hospital
        - there is a fully equipped team to re-evaluate the situation
        - the team will be able to support the parents and family members
        - it helps to secure the scene of death for evaluation later
- samples in the ED
    - no samples should be taken in obvious or suspected non-accidental injury

        - forensic samples should be taken by the home office pathologist
    - once death has been confirmed in the ED, the coroner assumes immediate responsibility for the body
    - blood samples taken during the attempted resuscitation can be sent for investigations
    - blood culture is a priority if small samples are obtained
    - all samples and the site from which they were taken should be documented
    - an unbroken chain of evidence should be secured
        - samples must be handed over to biochemistry/microbiology staff and must not be sent via a chute
    - urine or stool/stained nappy should be preserved and sent for analysis
    - supra-pubic puncture must not be done for urine samples
- photography of the child
    - a police photographer should take photos in suspicious deaths or suspected NAI
    - photographs of skin discolouration may be helpful to assess time of death/position in which the child was lying
    - photographs for bereavement purposes and locks of hair, footprints, handprints etc may be taken after parental consent
    - parents may take photos of their child using personal phones and cameras
- tubes and lines
    - IV/IO lines should be left in place but the tubing removed
    - chest drains/IV cannulas should be left in place but may be clamped
    - the ET tube must be left in place but may be cut off at the mouth and the cut end placed inside the cheek if possible
    - a complete documentation of all cannulation attempts, sites of venepuncture or IO needling, monitors and tubes should be made before their removal
    - in suspicious or criminal deaths the body must not be cleaned in any way
- care of parents and siblings
    - parents will be shocked and distraught – the emphasis is on sensitive, open-minded and balanced approach in dealing with the situation
    - immediately upon their arrival the parents should be allocated a member of staff to look after them, explain what is happening and provide facilities to contact friends, other family members and cultural or religious support
    - the paediatric SpR or ED SpR should, as part of the initial assessment, take a detailed and careful history of events leading up to and following the discovery of the infant's collapse
    - when the child has been pronounced dead, the ED team or on-call paediatric team should break the news to the parents, having reviewed all the available information; they must explain that possible medical causes will be very carefully and thoroughly sought; unless the cause of death is immediately apparent (e.g. RTC, meningococcal rash) it is important to explain that the cause of death is not yet known and that the aim of the investigation is to establish the cause of death
    - unexpected deaths:
        - strategic discussion involving the ED consultant and/or paediatric consultant on call with the police
        - enables sharing of information, identification of any initial concerns, clear division of responsibilities, plan for investigating the case and for giving information to and supporting the family

        - if there are child protection concerns a formal strategy discussion/meeting should be convened by Social Services under section 47 of the Children Act
    - parents and close relatives should be given the opportunity to hold and spend time with the child – always supervised by a member of nursing staff
    - the parents must be informed that the coroner will order a post-mortem examination in cases where a death certificate cannot be issued
        - they should be given and explanation and a leaflet
    - information leaflets on the child death review process should be provided
- SUDIC nurse
    - the named SUDIC nurse coordinates the CDOP process
    - a phase 2 meeting is arranged within 5-7 days (usually by phone)
        - ensures that all agencies are informed and updated, any concerns are identified and managed with an agreed action plan, including ongoing support to the family; the coroner is kept informed of all outcomes
    - a phase 3 meeting within 16-20 weeks once the PM is available
        - all relevant agencies are invited to share information and identify the cause of death, ongoing support to the family and any lessons learnt
- detailed history and examination (may be by the paediatric SpR or the ED SpR)
    - history
        - presenting history
            - record parent's account of events
            - ideally should be recorded verbatim
            - detailed history as per any critically ill child
        - basic details
            - of baby/child, parents and other family members (with family tree if possible)
        - narrative account of the 24 hours leading up to the child's death
            - in children <2, full description of:
                - when and how the baby slept and fed
                - any activity
                - who was with the baby at different times
                - baby's health and activity levels
                - final sleep
                - any changes to routine
                - where and how the baby was sleeping
                    - clothing
                    - bed coverings
                    - position
                - any changes during the night
                - if bed sharing, who else was in the bed and their positions in the bed relative to the baby
                - when and by whom the baby was checked during the sleep
                - description of the last feed and any night time feeds
                - heating and ventilation
            - in older children:
                - social issues
                - drug and alcohol use

        - mental health issues
    - where and how the child was found
        - position
        - coverings
        - appearance
        - any unusual features
        - any action taken after the child was found
    - past medical history
        - relevant to age
            - consider pregnancy and delivery
            - birth weight
            - post-natal problems
        - growth and development
        - normal routine and feeding
        - illnesses
        - immunisations
        - medications
        - drug allergies
        - routine surveillance
        - details of normal routine for the baby
            - feeding
            - sleeping patterns and practices
    - previous OPD, hospital, GP, HV and ED visits
    - family medical history
        - any medical or psychiatric history of the parents and other immediate family
        - infectious contacts
        - any history of respiratory, cardiac, neurological or metabolic disorders in the family
        - any previous infant or other sudden deaths in the family
        - if the child is a twin, the second twin MUST be examined and investigated by a paediatrician
    - social history
        - family structure and dynamics
        - housing
        - use of alcohol, recreational drugs and tobacco
        - parents' occupations
        - any social services involvement in the past including any child protection concerns
- examination
    - a detailed examination should be performed
    - a rectal temperature should be taken to assist the pathologist in estimating time of death
    - head to toe examination and from to back for bruising/injuries/visible signs of bleeding/discharge
        - use body diagrams to document the injuries
    - examination:

                - spine
                - skull
                - chest
                - upper limbs
                - lower limbs
                - genitalia
                - anal region
                - abdomen
                    - hepatomegaly
            - signs of dehydration
            - weight, length/height
            - state of nutrition and cleanliness
            - petechiae
            - eye examination
                - retinal haemorrhages may be difficult to see if the cornea is clouded but should be commented on if present
            - ENT exam
                - frenulum
                - bleeding
                - pink fluid from nose
                - presence of frothy fluid around the nose or mouth and its colour should be documented
            - sites of medical intervention (e.g. IV/IO lines) needs to be documented
            - presence and distribution of any discolouration of the skin, particularly dependent livido
                - skin livido and pallor from local pressure (e.g. on the nose in a patient who has been face down)
                - in older children signs of self-harm (e.g. cutting)

ALLERGY

AP1 acute allergy

- background
    - similar to anaphylaxis but does not meet all the requirements (e.g. just skin manifestations)
    - type I hypersensitivity reaction
        - IgE mediated
        - degranulation of mast cells and basophils
        - usually happens after a first exposure which causes sensitisation
- clinical features
    - presentation can be delayed
- differential
    - urticaria
    - anaphylaxis
    - angioedema
    - anxiety attack

    - asthma exacerbation
    - carcinoid syndrome
    - cold urticaria
    - contrast induced allergic reaction
    - scombroid
    - shock
    - transfusion reaction
- evaluation
    - clinical
        - rule out anaphylaxis
        - can be difficult to differentiate from angioedema
- management
    - H1 antagonist
        - e.g. diphenhydramine, fexofenadine, loratadine, cetirizine
    - H2 antagonist
        - e.g. ranitidine 150mg IV/IM/PO
            - improves urticaria but not angioedema at 2 hours
    - consider corticosteroid
        - e.g. prednisolone 60mg PO
        - can be continued as outpatient (40mg prednisolone PO for 5 days)

AP2 anaphylactoid reactions

- background
    - not IgE mediated but cause similar reactions
    - there is no detectable immunological sensitisation
- pathophysiology
    - mechanism is unclear
    - there is direct release of histamine caused by some substances such as opioids and contrast media
    - direct activation of the complement system and kinin-kallikrein system may play a role
    - local anaesthetics or stress can induce neuropsychogenic reflexes leading to release of mediators
- common precipitants
    - drugs (almost any can cause an anaphylactoid reaction)
        - lidocaine
        - prednisolone
    - food
        - peanuts
        - fish
        - gelatin
    - additives in drugs and food
    - occupational substances (e.g. latex)
    - animal venom (e.g. scorpion, snake)
    - aeroallergens
    - haemodialysis
    - contrast

    - seminal fluid
    - physical agents (e.g. cold, heat, UV irradiation)
    - hereditary angioedema
    - systemic mastocytosis
    - exercise
    - echinococcal cyst
    - idiopathic
- differential
    - vasovagal syncope
    - syncope
    - drug reaction
    - hypoglycaemia
    - seizure
    - foreign body/aspiration
    - psychogenic
    - pulmonary embolism
    - hyperventilation
- diagnosis
    - symptoms
        - clinical features similar to anaphylaxis but the presentation is almost always milder
        - usually begins with skin involvement
        - paraesthesia, itching of the pharynx and genital area and feeling of anxiety are common
        - almost all organ systems can be involved:
            - respiratory
                - sneezing/rhinorrhoea
                - hoarseness or throat tightness
                - cough and wheezing
                - dysphonia
                - dyspnoea due to laryngeal obstruction
                - cyanosis
                - respiratory arrest
            - gastrointestinal
                - nausea and cramping
                - vomiting and diarrhoea
                - micturition and defaecation
            - cardiovascular
                - tachycardia
                - hypotension
                - arrhythmia
                - shock
                - cardiac arrest
- grading
    - grade I
        - skin
            - pruritus

- flush
- urticaria
- angioedema

- grade II
  - skin
    - pruritus
    - flush
    - urticaria
    - angioedema
  - abdomen
    - nausea
    - cramping
  - respiratory
    - rhinorrhoea
    - hoarseness
    - dyspnoea
  - cardiovascular
    - tachycardia
    - arrhythmia
- grade III
  - skin
    - pruritus
    - flush
    - urticaria
    - angioedema
  - abdomen
    - vomiting
    - defaecation
    - diarrhoea
  - respiratory
    - laryngeal oedema
    - bronchospasm
    - cyanosis
  - circulatory
    - shock
- grade IV
  - skin
    - pruritus
    - flush
    - urticaria
    - angioedema
  - abdomen
    - vomiting
    - defaecation
    - diarrhoea
  - respiratory

                - respiratory arrest
            - cardiovascular
                - cardiac arrest
- management
    - treat as anaphylaxis

AP3 angioedema

- background
    - pathogenetically similar to urticaria but involves the deep dermal and subcutaneous tissue
    - aetiology can be either allergic (IgE and histamine mediated) or non-allergic
        - 90% are the allergic type
        - the non-allergic type includes:
            - drug-induced (e.g. ACE inhibitors)
            - hereditary (C1-esterase inhibitor deficiency)
            - acquired
            - idiopathic
            - pseudoallergenic
        - the non-allergic drug-induced, hereditary and acquired forms are mediated by bradykinin
    - drug reactions
        - those eliciting a type I hypersensitivity reaction produce angioedema within minutes
        - causing angioedema by inhibiting bradykinin may not be seen for months
        - the commonest drug classes associated with angioedema:
            - ACE inhibitors
            - bupropion
            - vaccines
            - SSRIs
            - COX-II inhibitors
            - angiotensin 2 antagonists
            - other antidepressants
            - NSAIDs
            - statins
            - PPIs
    - hereditary angioedema
        - an autosomal dominant condition caused by C1 esterase inhibitor deficiency or functional deficiency
        - can be confirmed clinically by low levels of C4 and C1 esterase inhibitor function
        - around 1 in 10,000 to 1 in 50,000 people are affected
- signs and symptoms
    - commonly involves swelling of the face, lips, mouth, tongue, extremities and genitalia
    - laryngeal involvement can produce stridor and lead to complete airway obstruction
    - associated abdominal symptoms caused by bowel wall oedema
        - colic-like pain, nausea, vomiting, diarrhoea

    - angioedema presents alone in around 10% of cases (should prompt consideration of non-allergic cause)
        - urticaria presents with angioedema in about 50% of cases
    - allergic angioedema
        - anatomically localised attack
        - urticaria
        - pruritus
        - normotension
    - non-allergic angioedema
        - anatomically localised attack
        - gradual onset
        - no pruritus
        - previous identical episodes
        - abdominal pain
        - normotension
    - anaphylaxis
        - systemic symptoms
        - rapid onset and progression
        - respiratory failure
            - wheeze
            - fatigue
            - cyanosis
            - hypoxia
            - tachypnoea
        - cardiovascular collapse
            - diaphoresis
            - hypotension
            - tachycardia
            - drowsiness
- management
    - mild allergic angioedema:
        - antihistamines are first line
    - moderate to severe allergic angioedema
        - consider IM adrenaline
            - treats peripheral vasodilatation and oedema formation (alpha adrenoceptors) and inhibits further mediator release (beta adrenoceptors)
        - intubation if there is threatened airway occlusion from laryngeal or oropharyngeal swelling
        - systemic corticosteroids
            - may be of use as an immune system modulator
            - take 6-8 hours to have an effect
            - may reduce the length of an attack
    - hereditary angioedema
        - does not usually respond to treatment with adrenaline, antihistamines and corticosteroids

        - should be treated with Icatibant (a bradykinin B2 receptor antagonist) or C1 esterase inhibitor concentrate if available
            - two units of fresh frozen plasma can be used as an alternative (contains C1 esterase inhibitor)
        - Icatibant is given as a single subcutaneous injection
            - produces symptomatic improvement in under an hour
        - C1 esterase inhibitor concentrate and FFP are derived from donated blood and require IV administration
- follow up
    - mild to moderate angioedema
        - if patients have only required treatment with antihistamines they can be discharged with no follow up
            - advice to avoid precipitant (if known)
            - advice to take oral antihistamines for further attacks
    - drug precipitants should be sought and discontinued if implicated
        - ACE inhibitors will need to be replaced with a different antihypertensive
    - patients requiring adrenaline or suspected of HAE
        - require follow up in allergy clinic for formal diagnosis
        - should be considered for an Epipen prescription
        - mast cell tryptase during an attack and convalescence may be helpful

AP4 urticaria

- background
    - an itchy red blotchy rash resulting from swelling of the superficial part of the skin
    - can be localised or more widespread
    - angioedema occurs when the deeper tissues, the lower dermis and subcutaneous tissues are involved and become swollen
- appearance
    - typically a central itchy white papule or plaque due to swelling of the surface of the skin (wheal) surrounded by an erythematous flare
    - lesions are variable in size and shape and may be associated with swelling of the soft tissues of the eyelids, lips, tongue (angioedema)
    - individual lesions are typically transient and come and go within a few minutes or hours
    - individual wheals may join to form larger patches
- classification
    - acute
        - symptoms develop quickly and resolve quickly (often within 48 hours)
        - more common in children, women, 30-60 year olds and people with atopy
    - chronic
        - rash persists for more than 6 weeks
- aetiology
    - activation of mast cells in the skin with release of histamine and other mediators
    - the chemicals cause capillary leakage, which causes swelling of the skin, and vasodilation causing the erythematous reaction
    - there may be an identifiable trigger but this is not always the case, particularly in chronic urticaria

        - an autoimmune reaction may be involved
- clinical classification
    - acute urticaria
        - cause identifiable in about half of cases
        - possible triggers include:
            - allergies
                - food
                - bites
                - stings
                - medication
            - viral infections
            - skin contact with chemicals, nettles, latex etc.
            - physical stimuli
                - firm rubbing (dermatographism)
                - pressure
                - cold
                - heat
    - chronic urticaria
        - chronic spontaneous urticaria
            - triggers include:
                - medication
                - stress
                - infection
            - previous called idiopathic urticaria
        - autoimmune urticaria
            - comes under chronic spontaneous urticaria in the European guidelines
            - may account for more than half of all cases of chronic urticaria in adults and older children
            - there may be an association with other autoimmune conditions
        - inducible urticaria
            - contact with hot or cold water (aquagenic)
            - exercise or emotion (cholinergic)
            - exposure to cold or heat
            - firm rubbing, minor trauma (dermatographism)
            - pressure (delayed pressure)
            - vibration
            - sun exposure (solar)
    - urticarial vasculitis
        - vasculitis of the skin characterised by inflammation of the small blood vessels
        - causes include infection (hepatitis B/C, glandular fever, streptococcal infection), medication (penicillins, fluoxetine, thiazides, allopurinol, quinolones, carbamazepine), autoimmune disease, paraproteinaemia, malignancy
- differential
    - erythema multiforme

    - dermatitis herpetiformis
    - pemphigoid
    - erysipelas
    - eczema
    - urticaria pigmentosa
    - chronic pruritus
    - polymorphic eruption of pregnancy
    - urticarial vasculitis
- investigation
    - diagnosis usually made clinically with no investigation
        - a detailed history may point to a trigger in some cases
    - in chronic or recurring cases, investigations are guided by history; may include:
        - FBC
        - ESR/CRP
        - physical challenge
            - cold provocation test (ice cube)
            - heat provocation test (warm water)
            - pressure testing
            - UV light testing
            - exercise or hot bath testing for cholinergic urticaria
        - elicit dermatographism
        - patch/prick testing for contact urticarias
        - IgE tests for specific allergens
        - thyroid autoantibodies if autoimmune mechanism suspected
        - exclusion of suspected medication or food
        - tests for infectious diseases
        - skin biopsy (urticarial vasculitis)
- management
    - identify and treat the cause where possible
    - minimise nonspecific aggravating factors such as overheating, stress, alcohol, caffeine, medication likely to cause urticaria
    - topical antipruritic agents such as calamine lotion or topical menthol 1% in aqueous cream may soothe symptoms
    - non-sedating antihistamines
        - usually cetirizine, loratadine, fexofenadine
        - once symptom control is achieved the antihistamine should be continued for 3-6 months
        - if ineffective, doses of up to four times the standard dose may be used or another antihistamine added
        - a sedating antihistamine such as chlorphenamine may be helpful if itch interferes with sleep
        - antihistamines should be avoided if possible in pregnancy (chlorphenamine is usually the first choice if unavoidable)
    - where symptoms are severe, a short course of steroids may be helpful (e.g. prednisolone 40mg for 7 days)
    - second line options that may be used in secondary care include:
        - antileukotrienes (e.g. montelukast) in combination with an H1 antihistamine

        - omalizumab, an anti IgE antibody
            - effective in 80%
            - requires monthly injections
            - relapse is common when it is stopped
        - ciclosporin for the immunosuppressant effect
- prognosis
    - variable
    - most cases of idiopathic urticaria resolve over six months
        - a minority can persist for years
        - some remit and relapse
        - 50% resolve within 3-5 years
    - at least 20% of cases of chronic urticaria patients requiring referral to secondary care are still symptomatic 10 years after first presentation
        - factors associated with long duration include: severe symptoms, associated angioedema, positive antithyroid antibodies
    - complications include insomnia, depression, poorer quality of life

AC1 drug allergy/severe adverse drug reactions

- background
    - any drug can cause a skin reaction, some are more associated with certain types of reaction
    - not all dermatological problems have visible signs and there may be marked pruritus with normal looking skin
    - many reactions are immunological in origin
        - the drug may act as a hapten and bind to proteins to create a structure that the immune system recognises as 'not self'
        - hypersensitivity classes:
            - type I
                - IgE mediated
                - urticaria, angioedema, anaphylaxis
                - often cause by proteins and especially insulin
            - type II
                - cytotoxic reaction
                - haemolysis and purpura
                - caused by penicillins, cephalosporins, sulphonamides and rifampin
            - type III
                - immune complex reactions
                - vasculitis, serum sickness, urticaria
                - caused by salicylates, chlorpromazine, suphonamides
            - type Iv
                - delayed type reactions with cell-mediated hypersensitivity
                - contact dermatitis, exanthematous reactions, photoallergic reactions
                - most common, usually caused by topical applications
                - not dose dependent
                - usually begin 1-3 weeks after treatment started

        - may recur if other chemically-related drugs used
- diagnosis
    - history
        - caution against accepting the patient's idea of what the cause is
        - all medications, including prescribed, OTC, alternative and illicit
        - foods such as strawberries, shellfish
        - viral infections
        - whether the patient has had the drug before and whether there was a reaction then
- allergic patterns identified by NICE
    - immediate
        - develops within 1 hour
        - anaphylaxis, urticaria, exacerbation of asthma (e.g. by exposure to NSAIDs)
    - non-immediate without systemic involvement
        - 6-10 days after first drug exposure or within 3 days of second exposure
        - widespread red macules or papules or fixed drug eruption
    - non-immediate reactions with systemic involvement
        - 2-6 weeks after first drug exposure or within 3 days of second drug exposure
        - drug reaction with eosinophilia and systemic symptoms (DRESS) or drug hypersensitivity reaction (DHS)
        - widespread red macules, papules or erythroderma, fever, lymphadenopathy, liver dysfunction, eosinophilia
    - toxic epidermolysis
        - 7-14 days after first drug exposure or within 3 days of second exposure
    - Stevens-Johnson syndrome
        - 7-14 days after first drug exposure or within 3 days of second exposure
    - acute generalised exanthematous pustulosis (AGEP)
        - 3-5 days after first drug exposure
        - widespread pustules, fever, neutrophilia
- associated drugs and rashes
    - acneform lesions
        - tend to be over the upper body rather than the face, no comedones
        - typical drugs are:
            - corticosteroids
            - halogens
            - haloperidol
            - hormones
            - isoniazid
            - lithium
            - phenytoin
            - trazodone
    - acute generalised exanthematous pustulosis (AGEP)
        - acute onset of fever and generalised scarlatiniform erythema with many small, sterile, non-follicular pustules; appears like pustular psoriasis
        - mostly caused by antibiotics
        - other causes include viral infections, mercury exposure, UV radiation
        - typical drugs include:

                - beta lactams
                - macrolides
                - paracetamol
                - carbamazepine
                - tetracyclines
                - diltiazem
                - furosemide
                - hydroxychloroquine
                - nifedipine
                - phenytoin
                - pseudoephedrine
                - ranitidine
                - sertraline
                - simvastatin
                - terbinafine
                - vancomycin
            - resolves spontaneously and rapidly with fever and pustules lasting 7-10 days before desquamation over a few days
    - alopecia
        - associated drugs include:
            - ACEi
            - allopurinol
            - anticoagulants
            - azathioprine
            - beta-blockers
            - hormones
            - NSAIDs
            - phenytoin
            - methotrexate
            - valproate
    - bullous pemphigoid
        - associated drugs include:
            - captopril
            - chloroquine
            - ciprofloxacin
            - furosemide
            - neuroleptics
            - penicillins
            - sulphasalazine
    - erythema nodosum
        - most common with oral contraceptives
        - other drugs include:
            - halogens
            - penicillin
            - sulphonamides
            - tetracyclines

- erythroderma
  - diffuse redness of the skin
  - drugs include:
    - allopurinol
    - anticonvulsants
    - captopril
    - chloroquine
    - diltiazem
    - lithium
    - nitrofurantoin
    - omeprazole
    - phenytoin
    - St John's wort
    - sulphonamides
- fixed drug eruptions
  - lesions recur in the same area when the same drug is given
  - plaques are circular, violaceous and oedematous and resolve with macular hyperpigmentation
  - latent period is half and hour to 8 hours after taking the drug
  - most usual sites are hands, feet and genitalia
- hypersensitivity syndromes
  - true allergy
  - drugs include:
    - allopurinol
    - amitriptyline
    - carbamazepine
    - lamotrigine
    - NSAIDs
    - olanzapine
    - phenytoin
    - spironolactone
- lichenoid reactions
  - lesions similar to lichen planus, may have marked pruritus
  - drugs include:
    - amlodipine
    - antimalarials
    - beta-blockers
    - diltiazem
    - furosemide
    - gold
    - PPIs
    - sildenafil
    - tetracycline
    - thiazides
- lupus drug reactions
  - produces symptoms like SLE but with uncommon skin findings or a drug-induced subacute cutaneous lupus erythematosus (SCLE) characterised by

annular psoriasiform, non-scarring lesions in a typical pattern for photosensitivity

- photosensitivity
  - drugs include:
    - ACEi
    - amiodarone
    - amlodipine
    - diltiazem
    - furosemide
    - nifedipine
    - quinolones
    - sulphonamides
    - tetracyclines
    - thiazides
- urticaria
  - most likely drugs:
    - bupropion
    - carbamazepine
    - chlordiazepoxide
    - fluoxetine
    - lamotrigine
    - lithium
    - trazodone
- vasculitis
  - most likely with:
    - fluoxetine
    - paroxetine
    - trazodone

- potentially fatal drug reactions
  - Stevens-Johnson syndrome and toxic epidermal necrolysis
    - often caused by infections, particularly herpes simplex
    - usually penicillins or sulphonamides when caused by drugs
  - features include:
    - initial fever, sore throat, chills, headache, arthralgia, vomiting, diarrhoea, malaise
    - lesions may occur anywhere but most commonly affect the palms, soles, dorsum of hands and extensor surfaces
      - rash may be confined to any one area of the body, usually the trunk
      - there is no pruritus
    - mouth involvement may be severe enough that the patient cannot eat or drink
    - genitourinary involvement may result in dysuria or inability to pass urine
    - rash can begin as macules that develop into papules, vesicles, bullae, urticarial plaques, confluent erythema
    - the typical lesion has the appearance of a target
    - the skin loss of TEN is best managed in a burns unit

        - both, but especially TEN, can result in scarring, severe sight impairment and even death
- investigations
    - usually none required
- common causes of non-immunological drug reactions
    - Jarisch-Herxheimer reaction
        - occurs on starting penicillin for syphilis due to the rapid destruction of spirochaetes and release of endotoxins
    - photosensitivity
        - caused by production of free radicals or reactive oxygen species
    - agyria
        - accumulation of silver from silver nitrate nasal sprays
        - causes blue-grey discolouration of skin and nails
- management
    - removal of the offending drug
        - if the condition resolves as expected a note of adverse reaction should be made
    - antihistamines for symptomatic relief unless part of the problem
    - corticosteroids for DRESS
- prognosis
    - most cases resolve without complications
    - rash may take 10-14 days to disappear
    - patients with exanthematous reactions will have mild desquamation as the rash resolves
    - Stevens Johnson syndrome has a mortality of around 10%, TEN 50%

CARDIOLOGY

CP1 chest pain

- causes
    - potentially life-threatening
        - cardiovascular
            - acute coronary syndromes
            - aortic dissection
            - myocarditis (most common cause of sudden death in the young)
            - pericarditis
            - pulmonary embolism
        - other
            - tension pneumothorax
            - acute chest syndrome (sickle cell disease)
            - Boerhaave syndrome
    - common non life-threatening causes
        - gastrointestinal
            - biliary colic
            - GORD
            - peptic ulcer disease

        - pulmonary
            - pneumonia
            - pleurisy
        - chest wall syndromes
            - musculoskeletal pain
            - costochondritis
            - thoracic radiculopathy
            - Bornholm disease
                - acute transient viral myositis associated with Coxsackie B
            - Tietze syndrome
                - idiopathic benign inflammation of one or more of the costal cartilages
            - Texidor twinge
                - precordial catch syndrome
            - Mondor disease
        - psychiatric
            - anxiety
            - somatisation
            - Da Costa syndrome
                - palpitations, dyspnoea, precordial pain, fatigue, exaggerated emotional responses with increased cardiac awareness
        - occult trauma
            - rib fractures
        - infection
            - shingles
- risk stratification for chest pain
    - HEART score
        - for undifferentiated chest pain
        - stratifies into low, moderate and high risk
            - identifies patients at higher risk of having a MACE (all-cause mortality, MI or coronary revascularisation) within the next 6 weeks
        - components:
            - **h**istory
                - slightly suspicious (0)
                - moderately suspicious (1)
                - highly suspicious (2)
            - **E**CG
                - normal (0)
                - non-specific repolarisation disturbance (1)
                    - includes LBBB, LVH, repolarisation changes
                - significant ST deviation (2)
                    - not due to LBBB, LVH or digoxin
            - **a**ge
                - <45 (0)
                - 45-64 (1)
                - ≥65 (2)
            - **r**isk factors

        - hypertension, hypercholesterolaemia, diabetes, obesity, smoking (current or within past 3 months), positive family history (parent or sibling with CVD before 65), atherosclerotic disease (prior MI, PCI, CABG, CVA/TIA, peripheral arterial disease)
        - no known risk factors (0)
        - 1-2 risk factors (1)
        - ≥3 risk factors or history of atherosclerotic disease (2)
    - **t**roponin (initial)
        - ≤ normal limit (0)
        - 1-3x normal limit (1)
        - >3x normal limit (2)
  - 0-3 – low score
  - 4-6 - moderate score
  - 7-10 – high score

CP2 breathlessness

- acute dyspnoea differential
    - emergent
        - pulmonary
            - airway obstruction
            - anaphylaxis
            - angioedema
            - aspiration
            - asthma
            - cor pulmonale
            - inhalation exposure
            - noncardiogenic pulmonary oedema
            - pneumonia
            - pneumocystis pneumonia
            - pulmonary embolism
            - pulmonary hypertension
            - tension pneumothorax
            - idiopathic pulmonary fibrosis (acute exacerbation)
            - cystic fibrosis (exacerbation)
        - cardiac
            - cardiac tamponade
            - cardiogenic pulmonary oedema
            - myocardial infarction
            - pericarditis
            - myocarditis
        - associated with normal/increased respiratory rate
            - abdominal distension
            - anaemia
            - carbon monoxide poisoning
            - salicylate toxicity
            - DKA

                - diaphragmatic injury
                - electrolyte abnormalities
                - epiglottitis
                - flail chest
                - hypotension
                - metabolic acidosis
                - pneumonia
                - pneumothorax/haemothorax
                - renal failure
                - sepsis
                - toxic ingestion
            - associated with decreased respiratory effort
                - Guillain-Barré syndrome
                - multiple sclerosis
                - myasthenia gravis
                - Lambert-Eaton syndrome
                - organophosphate toxicity
                - stroke
                - tick paralysis
    - non-emergent
        - ALS
        - ascites
        - uncorrected ASD
        - congenital heart disease
        - COPD exacerbation
        - fever
        - hyperventilation
        - interstitial lung disease
        - neoplasm
        - obesity
        - panic attack
        - pleural effusion
        - polymyositis
        - porphyria
        - pregnancy
        - rib fracture
        - spontaneous pneumothorax
        - thyroid disease
        - URTI
- investigations
    - may include:
        - CXR
        - ECG
        - WCC
        - U&Es
        - BNP
        - d-dimer

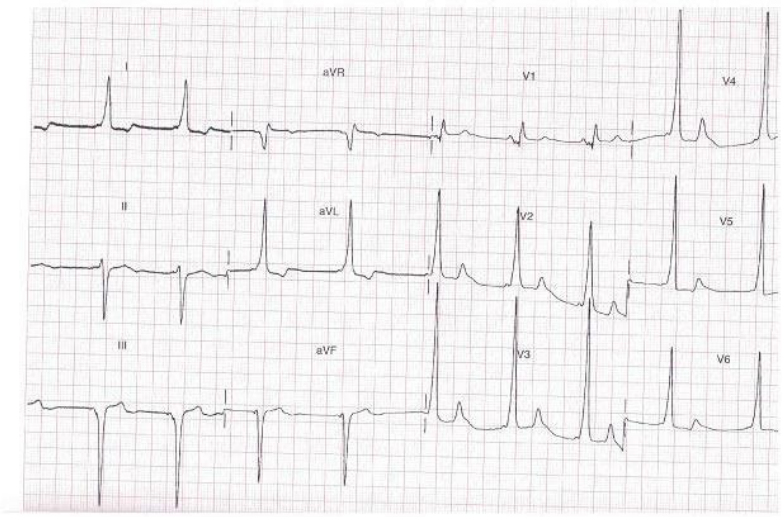
        - troponin
        - ABG
        - bedside ultrasound
            - predominant A lines + lung sliding – asthma/COPD
            - multiple predominant B lines anteriorly with lung sliding – pulmonary oedema
            - normal anterior profile + DVT – PE
            - anterior absent lung sliding + A lines + lung point – pneumothorax
            - anterior alveolar consolidations, anterior diffuse B lines with abolished lung sliding, anterior asymmetric interstitial patterns, posterior consolidations or effusions without anterior diffuse B lines - pneumonia

CP3 palpitations

- background
    - second commonest cardiac related presentation to ED
    - majority have excellent prognosis and the challenge is to identify the minority with significant dysrhythmia or underlying disease
    - clarification of symptoms is important
        - usually described as increased awareness of own heartbeat
        - may be fast or irregular heartbeat or excessive pulsation
    - usually unpleasant or distressing, which leads to seeking medical help
    - more likely to be tachyarrhythmia than bradyarrhythmia
        - change of rate, rhythm or contractility probably alters the usual physiological state in which there is a filtering of afferent feedback so cardiac activity is subconscious
- pathophysiology
    - cardiac
        - arrhythmia
            - sinus arrhythmia
            - atrial fibrillation
            - atrial flutter
            - heart block
            - atrial tachycardia
            - SVT
            - VT
            - pre-excitation (WPW)
            - PVC
        - non-arrhythmic
            - sinus tachycardia
            - congenital structural disease
            - acquired valvular disease
            - heart failure
            - cardiomyopathy
            - pericarditis
            - myocarditis
    - non-cardiac

        - drugs/toxins
            - sympathomimetics
            - cardiac drugs
            - illicit drugs
            - alcohol
            - nicotine
            - caffeine
            - serotonergic drugs
            - sodium channel blockers
            - therapeutic drug withdrawal
        - systemic
            - anaemia
            - metabolic disorder
            - pyrexia
            - hypovolaemia
            - hyperthyroidism
            - phaeochromocytoma
            - pulmonary disease
            - hypoxia
        - psychiatric
            - anxiety disorder
            - somatisation
    - underlying cardiac cause found in 43%, anxiety in 31%, no specific cause in 16%
- history
    - exact nature of palpitations
        - rate
        - rhythm
        - missed beat sensation
        - extra beat sensation
        - duration
        - frequency
        - precipitating factors (including dietary, posture, exercise and sleep)
        - relieving factors
    - presence of adverse clinical features
        - chest pain
        - dyspnoea
        - sweating
        - dizziness
        - syncope
        - extreme fatigue
    - presence of symptoms which may indicate an underlying cause
        - chest pain (in the absence of or preceding palpitations)
        - tremor
        - sweating
        - abdominal pain
        - anxiety
        - heat intolerance

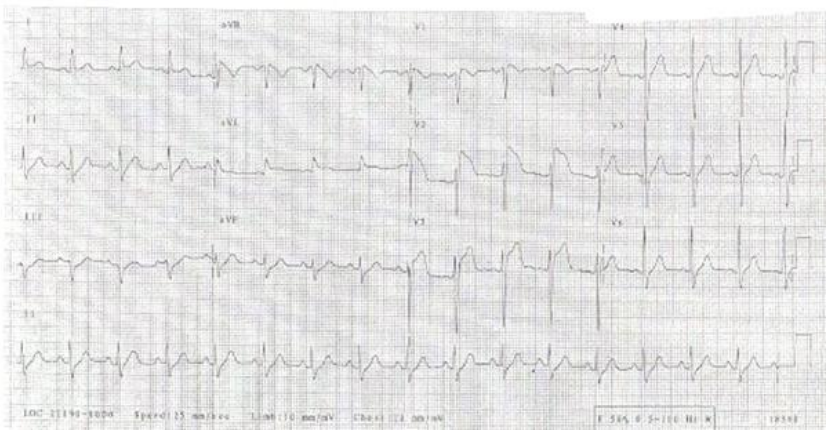
        - weight change
        - productive cough
        - depression
        - weakness
        - fatigue
    - past medical, drug and social history
        - history of structural heart disease
        - family history of sudden cardiac death, particularly in first degree relatives
        - prescription and over the counter medications
            - including herbal or alternative preparations
        - alcohol and illicit drug use
- examination
    - full cardiovascular examination
        - rate and rhythm
        - auscultation of heart
        - signs of heart failure
        - murmurs
        - altered apex beat
        - cardiovascular collapse
    - signs of systemic illness
        - pyrexia
        - pallor
        - thyrotoxic features
        - signs of drug or alcohol intoxication
    - brief psychological assessment
        - to assess for heightened anxiety
- investigations
    - 12 lead ECG
        - high yield if recorded at time of palpitations
        - in absence of palpitations at the time, look for:
            - pre-excitation (e.g. WPW)
                - may suggest recurrent SVT



ECG showing WPW

https://www.rcemlearning.co.uk/reference/palpitations/#1570787252268-9bafd3af-bbf0

- p wave abnormalities
- left ventricular hypertrophy
- abnormal QRST morphology (e.g. Brugada syndrome)



https://www.rcemlearning.co.uk/reference/palpitations/#1570787252268-9bafd3af-bbf0

- frequent ventricular premature beats
- Q waves
- abnormal QT duration (long and short)
- atrioventricular block
    - may cause symptomatic bradyarrhythmias
- fascicular block

- bloods
    - U&Es
    - calcium
    - magnesium
    - FBC
    - TFTs

        - glucose
- ED management
    - is intervention required?
        - manage arrythmias as per guidelines
        - management of any specific underlying cause identified
    - is admission required?
        - imminent risk of life-threatening arrhythmia (e.g. previously recorded episode of VT)
        - adverse symptoms and signs present during the palpitations
        - implanted cardiac devices suspected of malfunction
        - family history of sudden death (e.g. Brugada syndrome)
        - admission required for investigation or treatment of underlying cause or precipitating illness
        - admission not required if innocent diagnosis confidently achieved or symptoms have resolved, no specific aetiology identified and no high risk features
    - is further investigation required if diagnosis not made?
        - referral to cardiology:
            - suspected arrhythmia or structural heart disease without high risk features mandating admission
        - options for those without a suspected or confirmed specific diagnosis include:
            - referral to GP for follow up and discretionary cardiology referral
                - suitable for low risk patients who report occasional missed beats
            - referral directly to cardiology for outpatient follow up
            - referral for ambulatory monitoring +/- echocardiography and subsequent cardiology follow up
                - outpatient echo should be organised directly in patients suspected of structural heart disease
- discharge advice
    - avoidance of stimulants such as alcohol, caffeine, nicotine
    - advise to keep a record of any episodes and associated symptoms
    - advise to reattend ED if palpitations recur and are persistent or associated with adverse features
    - if SVT suspected, patient should be taught Valsalva manoeuvre to attempt self-termination
    - anyone with a concerning cause of palpitations should be advised to exercise at light to moderate intensity pending cardiology review

CP4 transient loss of consciousness

- background
    - syncope is transient, self-limited loss of consciousness with an ability to maintain postural tone that is followed by spontaneous recovery
        - an event without loss of consciousness is termed presyncope
    - the underlying cause is not found in the ED in around 50%

        - cause is mostly found from history and examination rather than investigations
    - about 3-5% of ED visits
    - cardiac causes are more common in the elderly and those with a history of cardiac disease and they have the highest mortality
- causes
    - neurally-mediated reflex syncopal syndromes
        - vasovagal faint
        - carotid sinus syncope
        - situational faint
            - acute haemorrhage
            - cough, sneeze
            - gastrointestinal stimulation
                - swallow
                - defaecation
                - visceral pain
            - micturition
            - post-exercise
            - others
                - e.g. brass-instrument playing, weightlifting, post-prandial
        - glossopharyngeal and trigeminal neuralgia
    - orthostatic
        - autonomic failure
            - primary autonomic failure syndromes
                - pure autonomic failure
                - multiple system atrophy
                - Parkinson's disease with autonomic failure
            - secondary autonomic failure syndromes
                - diabetic neuropathy
                - amyloid neuropathy
            - drugs and alcohol
        - volume depletion
            - haemorrhage
            - diarrhoea
            - Addison's disease
    - cardiac arrythmias as primary causes
        - sinus node dysfunction
            - including bradycardia/tachycardia syndrome
        - atrioventricular conduction system disease
        - paroxysmal supraventricular and ventricular tachycardias
        - inherited syndromes
            - long/short QT syndrome
            - Brugada syndrome
            - WPW
            - arrhythmogenic right ventricular cardiomyopathy
        - implanted device malfunction
            - ICD, PPM

        - drug-induced arrhythmias
    - structural cardiac or cardiopulmonary disease
        - cardiac valvular disease
            - e.g. aortic stenosis
        - acute myocardial infarction/ischaemia
            - e.g. RV infarction
        - obstructive cardiomyopathy
        - atrial myxoma
        - acute aortic dissection
        - pericardial disease/tamponade
        - pulmonary embolism/pulmonary hypertension
    - cerebrovascular
        - vascular steal syndromes
- differential of syncope
    - disorders with impairment or loss of consciousness
        - metabolic disorders
            - hypoglycaemia (not true syncope as not spontaneously reversible)
            - hypoxia
            - hyperventilation with hypocapnia
        - epilepsy
        - intoxications
        - vertebro-basilar transient ischaemic attack
    - disorders resembling syncope without loss of consciousness
        - cataplexy
        - drop attacks
        - psychogenic 'syncope' (somatisation disorders)
        - transient ischaemic attack of carotid origin
- mnemonic (HEAD, HEART, VESSELS)
    - CNS causes (HEAD)
        - **h**ypoxia
        - **e**pilepsy (not a true cause of syncope)
        - **a**nxiety and hyperventilation
        - **d**ysfunctional brain stem (vertebrobasilar TIA)
    - cardiac causes (HEART)
        - **h**eart attack (ACS)
        - **e**mbolism (PE)
        - **a**ortic obstruction (e.g. AS, myxoma)
        - **r**hythm disturbance, ventricular
        - **t**achycardia
    - vascular causes (VESSELS)
        - **v**asovagal (emotional) or Valsalva (micturition, cough, straining etc.)
        - **e**ctopic (and other causes of hypovolaemia)
        - **s**ituational
        - **s**ubclavian steal
        - **E**NT (glossopharyngeal neuralgia)
        - **l**ow systemic vascular resistance
            - autonomic dysfunction (Addison's, diabetic vascular neuropathy)

- drugs such as calcium-channel blockers, beta blockers, antihypertensives
- **s**ensitive carotid sinus

- clinical assessment
  - circumstances just prior to attack
    - position
      - supine
      - standing
      - sitting
    - activity
      - rest
      - change in posture
      - during or after exercise
      - during or immediately after urination, defaecation, coughing, swallowing
    - predisposing factors
      - crowded or warm places
      - prolonged standing
      - post-prandial
    - precipitating events
      - fear
      - intense pain
      - neck movements
  - onset of attack
    - nausea
    - vomiting
    - abdominal discomfort
    - feeling of cold
    - sweating
    - aura
    - pain in the neck or shoulders
    - blurred vision
  - nature of the attack (eyewitness)
    - type of collapse
      - slumping, keeling over
    - skin colour
      - pallor
      - cyanosis
    - duration of loss of consciousness
    - breathing pattern
      - any snoring
    - movements
      - type
        - tonic
        - clonic
        - tonic-clonic
        - minimal myoclonus

        - automatism
      - duration
      - onset of movement in relation to fall
      - tongue-biting
  - end of attack
    - nausea
    - vomiting
    - sweating
    - feeling of cold
    - confusion
    - muscle aches
    - skin colour
    - injury
    - chest pain
    - palpitations
    - urinary or faecal incontinence
  - antecedents
    - family history of sudden death, congenital arrhythmogenic cardiac disease or fainting
    - previous cardiac disease
      - check for scars, pulses, murmurs, pacemakers and other devices
    - neurological history
      - Parkinsonism
      - epilepsy
      - narcolepsy
    - metabolic disorders, e.g. diabetes
    - medications
      - antihypertensives
      - antianginals
      - antidepressants
      - antiarrhythmics
      - diuretics
      - QT prolonging agents
    - recurrences – number of spells, time from first episode
  - examination
    - hydration status
    - pallor
    - evidence of blood loss
    - injuries due to collapse
    - postural drop
      - systolic <90mmHg or drop of >25mmHg on standing
      - reflex tachycardia
        - presence suggests hypovolaemia
        - absence suggests autonomic dysfunction
      - reproduction of symptoms is more important than numbers
    - rectal examination for melaena
- investigations

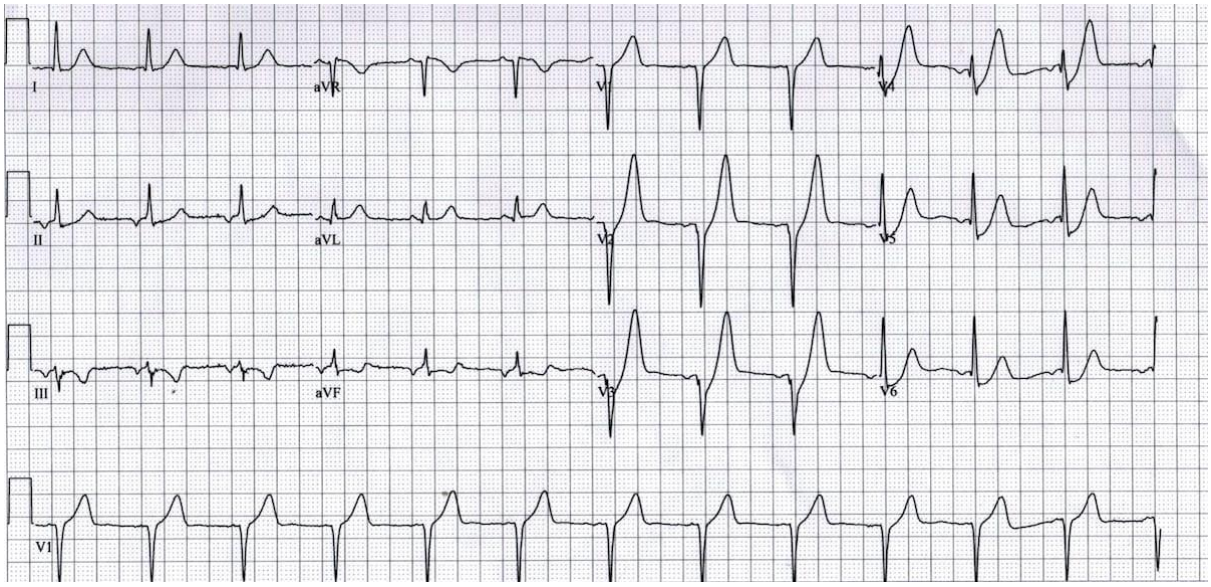
- depending on history and examination
  - labs
    - FBC (anaemia)
    - U&Es
    - glucose
    - VBG
    - d-dimer if PE suspected
    - consider blood alcohol
  - ECG
    - for causes of sudden collapse
      - **C**an **Q**uick **BRAD** **W**alk **H**ome
        - **c**onduction blocks
        - **Q**T – long/short
        - **B**rugada
        - **R**V infarction
        - **A**RVD (arrhythmogenic right ventricular dysplasia)
        - **D**CM (dilated cardiomyopathy)
        - **W**PW
        - **h**ypertrophy (HCM or LVH due to AS)
  - CT brain
    - if suspected first seizure
    - if secondary trauma suspected during syncopal episode
    - suspected TIA or stroke
    - neurological deficit or ongoing altered consciousness state/confusion
    - consider if age >65
    - sudden onset headache
    - patients on warfarin
- management
  - seek and treat underlying causes
  - lower threshold for admission:
    - syncope unwitnessed
    - significant risk factors including:
      - cardiovascular disease
      - documented or suspected arrhythmias
      - known epileptic with >1 seizure or without home supervision
      - cardiac pacemaker or other devices
    - elderly
    - suspected cardiac cause
      - monitoring and cardiology review
      - need to rule out ischaemic event and/or arrhythmias
      - high risk factors for cardiac cause include:
        - age
        - known electrophysiological abnormalities or previously documented malignant arrhythmias
        - diabetes
        - newly abnormal ECG

                - elevated troponin
                - significant depression of ventricular function on echo
                - documented IHD
                    - including past STEMI/NSTEMI, abnormal cardiac functional study, abnormal angiogram
                - pacemaker or other cardiac device
                    - require admission until device can be checked
            - suspected drug related cause
                - require drug review and observation
            - vasovagal
                - consider admission in the elderly or those with significant comorbidities
                - particularly if:
                    - recurrent episodes
                    - significant injuries have occurred
                    - lack of supervision at home
- discharge criteria
    - no significant risk factors:
        - abnormal ECG
        - CVS risk factors
        - initial hypotension
        - initial history of shortness of breath
    - witnessed seizure activity or history of seizures
    - observations and clinical findings are normal
    - medications reviewed
    - safe home environment

CC1 acute coronary syndromes

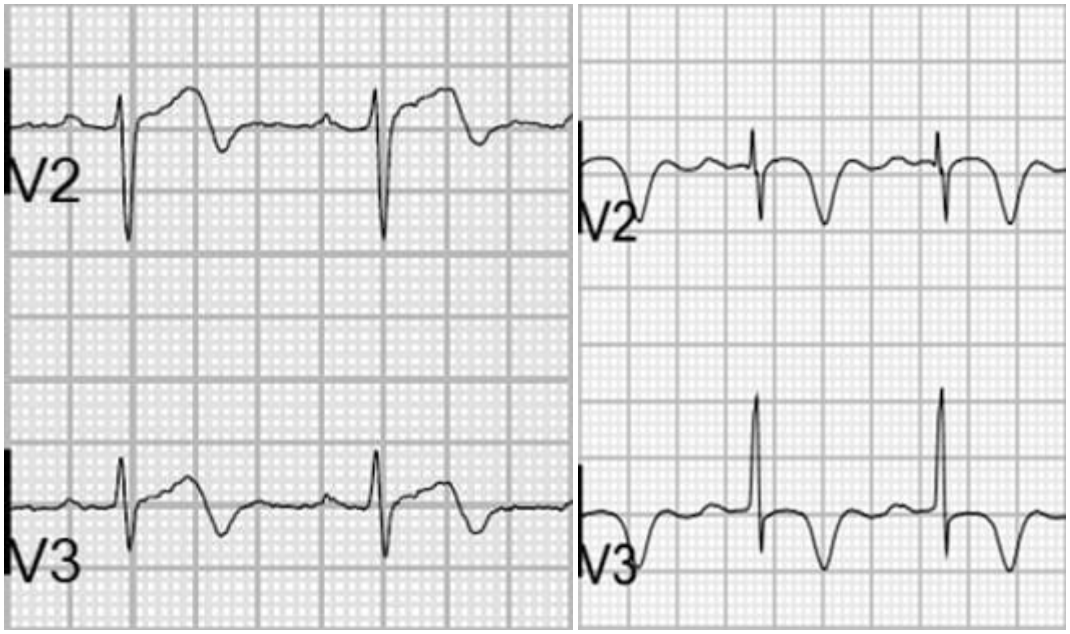
- background
    - coronary artery disease accounts for >30% of death in the West
    - ACS is a catch all term referring to ischaemic symptoms resulting from acute coronary occlusion
    - unstable angina is measured against a patient's normal pattern of stable angina; new onset angina should be considered unstable in the first instance
- risk factors for coronary artery disease
    - diabetes
    - hypertension
    - lipids
    - family history
    - male
    - obesity
    - previous MI
    - hormone replacement for menopause
    - inactivity
- New York Heart Association (NYHA) functional classification of angina
    - class I

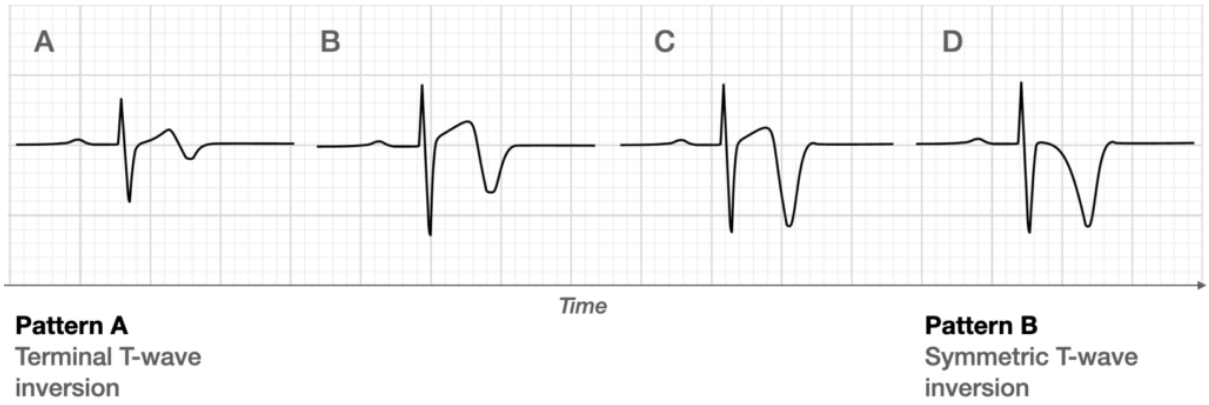
        - patients with cardiac disease but without resulting limitation of physical activity
        - ordinary physical activity does not cause undue fatigue, palpitation, dyspnoea or anginal pain
    - class II
        - patients with cardiac disease resulting in slight limitation of physical activity
        - comfortable at rest
        - ordinary physical activity results in fatigue, palpitation, dyspnoea or anginal pain
    - class III
        - patients with cardiac disease resulting in marked limitation of physical activity
        - comfortable at rest
        - less than ordinary activity results in fatigue, palpitation, dyspnoea or anginal pain
    - class IV
        - patients with cardiac disease resulting in inability to carry out any physical activity without discomfort
        - symptoms of heart failure or angina may be present even at rest
        - if any physical activity is undertaken, discomfort is increased
- risk stratification of patients with confirmed ACS
    - very high risk
        - haemodynamic instability
            - heart failure/cardiogenic shock
            - mechanical complications of myocardial infarction
        - life-threatening arrhythmias or cardiac arrest
        - recurrent or ongoing ischaemia (e.g. chest pain refractory to medical treatment) or recurrent dynamic ST segment and/or T wave changes, particularly with:
            - intermittent ST segment elevation
            - de Winter T wave changes
                - tall prominent symmetrical T waves in the precordial leads
                - upsloping ST segment depression >1mm at the J-point in the precordial leads
                - absence of ST elevation in the precordial leads
                - reciprocal ST segment elevation (0.5mm-1mm) in aVR
                - may be preceded or followed by ‘normal’ STEMI morphology
                - signifies proximal LAD occlusion and is equivalent to an anterior STEMI



https://litfl.com/wp-content/uploads/2018/08/ECG-De-Winter-T-Waves-1.jpg

- Wellens syndrome
    - pattern of deeply inverted or biphasic T waves in V2-3
    - highly specific for a critical stenosis of the LAD
    - patient may be pain free but is at extremely high risk of an extensive anterior wall MI in days to weeks
    - diagnostic criteria:
        - deeply inverted or biphasic T waves in V2-3 (may extend to V1-6)
        - isoelectric or minimally elevated ST segment (<1mm)
        - no precordial Q waves
        - preserved precordial R wave progression
        - recent history of angina
        - ECG pattern present in pain-free state
        - normal or slightly elevated serum cardiac markers





https://litfl.com/wellens-syndrome-ecg-library/

- widespread ST elevation in two or more coronary territories

- high risk
  - rise and/or fall in troponin level consistent with myocardial infarction
  - dynamic episode of ST segment and/or T wave changes with or without symptoms
  - GRACE score >140
- intermediate risk
  - diabetes
  - renal insufficiency (eGFR <60)
  - left ventricular ejection fraction ≤40%
  - prior revascularisation
    - percutaneous coronary intervention
    - coronary artery bypass grafting
    - GRACE score between 109 and 140
- low risk
  - no recurrent symptoms
  - no risk criteria

- risk stratification for ACS for patients presenting with chest pain (possible but not confirmed ACS)
    - high risk
        - clinical symptoms consistent with ACS and any of the following high risk features:
            - persistent or dynamic ST changes on ECG:
                - ST segment depression ≥ 0.5mm
                - new T wave inversion ≥ 2.0mm in more than 2 contiguous leads
                - transient ST segment elevation ≥ 0.5mm in more than 2 contiguous leads
                - ECG changes consistent with critical coronary artery stenosis
            - haemodynamic compromise
                - systolic blood pressure ≤90 mmHg
                - signs of poor peripheral perfusion
                - signs of cardiac failure or frank pulmonary oedema
                - new onset of mitral regurgitation
                - syncope
        - ongoing pain or recurrent episodes of pain despite initial treatment in ED
        - arrhythmias requiring treatment such as sustained VT
        - diaphoresis
        - elevated troponin
        - high risk co-morbidities
            - left ventricular systolic dysfunction
            - past PCI or CABG
            - past myocardial infarction
    - intermediate risk
        - those presenting with symptoms of ACS who do not have high or low risk criteria
    - low risk
        - age <40
        - symptoms that are atypical for angina
        - remain symptom free
        - absence of known CAD
        - normal troponin levels
        - normal ECG (including no transient changes)
- history
    - history of event
        - when pain started
        - nature and duration of pain
        - chest pain surrogates such as shortness of breath/lethargy
    - previous coronary interventions or investigations
    - comorbidities (including contraindications to thrombolysis)
    - medications
        - warfarin, antiplatelets or DOACs
        - has aspirin or another antiplatelet agent already been taken?
        - sildenafil (GTN may have an excessive effect if used)

    - allergies
        - especially aspirin
- examination
    - generally unhelpful in establishing ACS diagnosis
    - any immediate ABC issues
    - blood pressure
    - degree of anxiety/distress
    - any evidence of heart failure including S3/4, peripheral oedema, distended tender liver
    - evidence of cardiogenic shock (altered mental state, poor skin perfusion, hypotension)
- investigations
    - bloods
        - FBC
        - U&Es
        - glucose
        - troponin
            - rise may persist for 5-14 days post infarction
            - a falling pattern may be significant in patients who present late (already past the peak)
            - all patients with suspected ACS should have an initial troponin and then a repeat
    - ECG
        - patients with suspected ACS should have an ECG within 10 minutes of first clinical contact
        - initial decision is whether there is a STEMI
        - findings in ACS
            - may be normal
            - classic changes in acute MI
                - peaked T waves with ST elevation
                - gradual loss of R waves
                - development of pathological Q wave and TWI
    - CXR
        - cardiomegaly
        - cardiac failure
        - differentials for chest pain
            - PE, pneumonia, pneumothorax, oesophageal rupture, aortic dissection
    - echo
        - not a routine test but may be used for severe cases
        - confirmation of wall motion abnormalities
        - cardiogenic shock
        - secondary complications such as cardiac tamponade or valvular disruption
    - coronary angiography
        - definitive investigation for STEMI
        - should be considered for very high risk or high risk patients
- management

- initial management
  - oxygen therapy
    - to maintain sats >93% or if patient shocked
- analgesia
  - nitrates
    - sublingual, topical or as an IV infusion for intractable pain not responding to morphine
    - GTN 400 microgram sublingual, repeated every 5 minutes as needed to a maximum of 3 doses
  - opiates
    - e.g. IV morphine boluses titrated to clinical effect
      - incremental doses of 2.5-5mg
        - less in elderly or those with cardiorespiratory compromise
- anti-emetic as required
- antiplatelet therapy
  - for all patients with possible ACS and without genuine contraindications
  - 300mg aspirin dissolved or chewed as soon as possible after presentation
    - then 150mg daily continued indefinitely unless not tolerated or an indication for anticoagulation becomes apparent
  - other NSAIDs should not be given – increased risk of major adverse cardiac event (MACE)
  - P2Y12 inhibitor also recommended for patients with confirmed ACS and intermediate to high risk of recurrent ischaemic events
    - ticagrelor 180mg loading dose then 90mg BD
    - careful assessment of bleeding risk first
    - avoid if emergency CABG may be required
    - dual antiplatelet therapy (ticagrelor and clopidogrel) for up to 12 months in patients with confirmed ACS
- anticoagulation
  - recommended in patients with ACS at intermediate to high risk of ischaemic events
    - enoxaparin 1mg/kg SC (reduced for elderly or renal impairment)
- secondary prevention medications
  - statins
    - continued indefinitely after hospitalisation with ACS at highest tolerated dose
  - beta blocker
    - for patients with reduced left ventricular systolic function (ejection fraction <40%) unless contraindicated
  - ACEi/sartan
    - for patients with evidence of heart failure, left ventricular systolic dysfunction, diabetes, anterior MI or co-existent hypertension

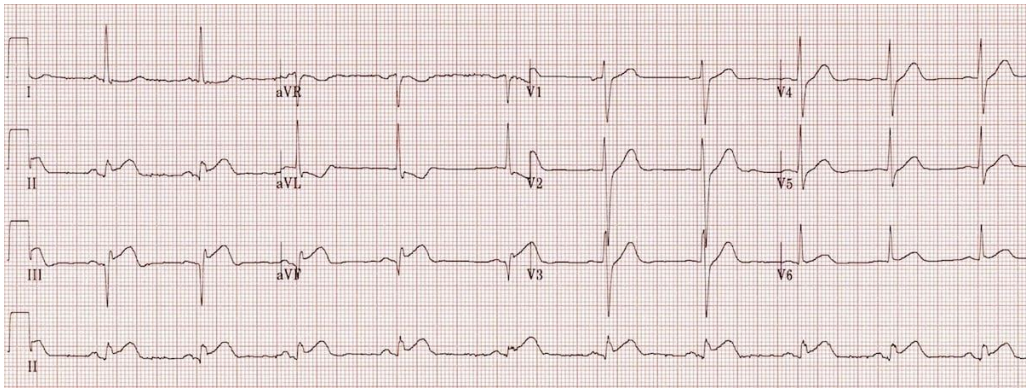
- disposition
  - high risk patients
    - all require hospital admission, most to CCU
    - criteria for coronary care admission includes:

                - significantly elevated serum troponin
                - recurrent chest pain whilst in the ED
                - ECG changes
                - haemodynamic instability
                - arrhythmias
            - patients require further objective testing during the admission
        - intermediate risk patients
            - non-invasive objective testing is recommended
            - may be accelerated for inpatient testing or discharged for outpatient testing within 7-14 days
        - low risk patients
            - no further objective testing for CAD recommended
- complications of acute coronary syndromes
    - cardiac failure
    - post-infarction ischaemia
    - ventricular free wall rupture
        - requires pericardiocentesis and repair
    - ventricular septal rupture
        - requires IABP, inotropes, surgery
    - acute mitral regurgitation
        - requires afterload reduction, IABP, inotropes, surgery ASAP
    - right ventricular infarction
        - requires IV fluids, inotropes, AV synchrony, IABP, reperfusion
    - arrhythmias
        - correct hypoxia, acidosis, hypovolaemia, potassium, magnesium
    - cardiogenic shock
        - must have revascularisation (PCI or CABG) within 24 hours
    - thromboembolism
        - anticoagulation for mural thrombus
    - pericarditis and Dressler's syndrome
    - complications of therapy
        - e.g. haemorrhage, coronary artery dissection, stent thrombosis, surgical complications

CC2 myocardial infarction

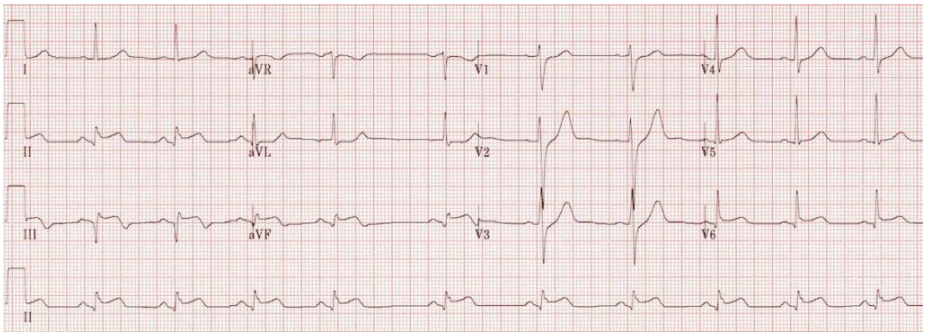
- definition
    - myocardial cell death following prolonged ischaemia
- universal classification of myocardial infarction
    - type 1
        - spontaneous MI
        - related to atherosclerotic plaque rupture, ulceration, erosion or dissection with resulting intraluminal thrombus in one or more of the coronary arteries
        - leads to decreased myocardial blood flow or distal platelet emboli with ensuing myocyte necrosis
    - type 2
        - MI secondary to ischaemic imbalance

        - myocardial injury with necrosis where a condition other than CAD contributes to an imbalance between myocardial oxygen supply and demand
            - e.g. coronary endothelial dysfunction, coronary artery spasm, coronary embolism, tachy/bradyarrhythmias, anaemia, respiratory failure, hypotension, hypertension with or without LVH
    - type 3
        - MI resulting in death when biomarkers are unavailable
        - cardiac death with symptoms suggestive of myocardial ischaemia and presumed new ischaemic ECG changes or LBBB but death occurred before blood samples could be obtained, before cardiac biomarkers could rise or where cardiac biomarkers were not collected
    - type 4a
        - MI related to PCI (complex specific criteria)
    - type 4b
        - MI related to stent thrombosis (complex specific criteria)
    - type 5
        - MI related to CABG (complex specific criteria)
- anatomical localisation of ST elevation
    - RCA
        - inferior MI
            - ST elevation II, III, aVF
            - may be preceded by hyperacute T waves
            - reciprocal ST depression aVL
            - usually dominant right circumflex artery (80%); 18% have dominant left circumflex



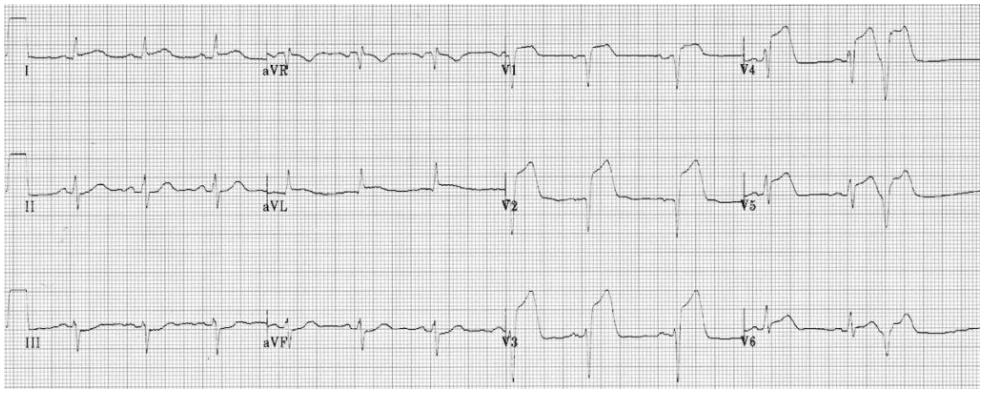
https://litfl.com/wp-content/uploads/2018/08/ECG-Inferior-AMI-STEMI.jpg

        - inferolateral
            - II, III, aVF, I, aVL, V5, V6



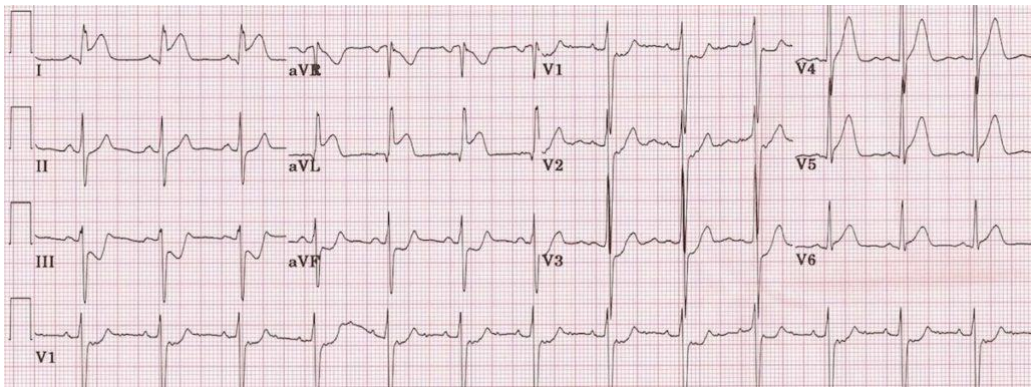
https://litfl.com/inferior-stemi-ecg-library/

- LAD
  - septal
    - V1, V2
  - anterior
    - elevation in precordial leads (V1-V6)
    - reciprocal depression inferior leads (III, aVF)



https://litfl.com/anterior-myocardial-infarction-ecg-library/

  - lateral
    - ST elevation I, aVL V5, V6
    - reciprocal ST depression inferior leads (III, aVF)
    - can be anterolateral (LAD occlusion); inferior-posterior-lateral, isolated lateral



https://litfl.com/lateral-stemi-ecg-library/

- STEMI

- presentation
  - chest pain due to imbalance between supply and demand of oxygen to the myocardium
  - demand increased when heart is working harder, supply decreased by occlusion of the coronary arteries
- management
  - simple measures to increase supply and reduce demand
    - supplementary oxygen if reduced sats
    - GTN
    - relief of pain and anxiety
      - these contribute to sympathetic overactivity, causing vasoconstriction and increasing cardiac workload
      - IV opiates drug of choice
      - beta blockers can be considered
        - 2.5-10mg bisoprolol PO
      - antiemetics
- PPCI
  - definition
    - angioplasty or stenting without prior or concomitant thrombolytic therapy
    - achieves and maintains coronary artery patency without exposing patients to the increased bleeding risks of thrombolysis
      - has better short-term mortality rate, reduced risk of re-infarction and lower incidence of stroke
    - thrombolysis generally preferred:
      - when there will be a delay to invasive strategy
        - when PCI not available within 2 hours from first medical contact or less than 90 minutes in early presenters (<2 hours post onset of symptoms)
    - PPCI is generally preferred:
      - if a skilled PCI laboratory is available
        - time from first medical contact to balloon inflation should be <2 hours in any case and <90 minutes in early presenters (<2 hours post onset of symptoms)
        - in patients with cardiogenic shock or a contraindication to thrombolysis
- thrombolysis
  - rarely used unless geographically distant to PPCI centre
  - can be administered pre-hospital by paramedics (bolus agents such as reteplase or tenectaplase)
  - risk factors for intracranial haemorrhage after thrombolysis are female gender, advanced age, low body weight and elevated blood pressure at presentation
  - contra-indications to thrombolysis
    - absolute
      - haemorrhagic stroke at any time
      - ischaemic stroke within 6 months

        - recent major surgery (within 3 weeks)
        - recent major trauma/head injury (within 3 weeks)
        - recent gastro-intestinal bleeding (within 1 month)
        - aortic dissection
        - known bleeding disorder
        - neurological deficit or CNS neoplasm
    - relative
        - warfarin therapy (check INR)
            - risk/benefit decision required based on INR
        - pregnancy or immediate post-partum period
        - TIA in preceding 6 months
        - prolonged or traumatic resuscitation
        - systolic BP >180mmHg
        - active peptic ulcer
        - advanced hepatic disease
        - non-compressible puncture site
        - infective endocarditis
- limitations to thrombolysis
    - failure to reperfuse
        - estimated to occur in up to 30%
        - patients should be referred for rescue PCI
    - thrombolysis should not be considered definitive treatment even if successful
        - pre-discharge angiography (within 6-24 hours) should be done
- adjunctive anti-thrombotic therapy
    - aspirin
        - irreversibly acetylates platelet cyclo-oxygenase and inhibits platelet aggregation
            - also an indirect antithrombotic agent
        - should be given early to all patients with ischaemic cardiac chest pain if no allergy or active GI bleeding
        - should be continued indefinitely
    - clopidogrel
        - promotes formation of platelet c-AMP, lowering platelet calcium and reducing platelet aggregation
    - prasugrel
        - acts in a similar way to clopidogrel but with faster onset
        - used for patients having PPCI
    - ticagrelor
        - novel oral antiplatelet agent
        - antagonist to P2Y12 ADP receptor
        - indicated to be used alongside aspirin in patients with acute coronary syndromes
    - heparin
        - prevents conversion of fibrinogen to fibrin, inhibiting clot formation
        - LMWHs work in a similar way and also bind to factor Xa

        - fondaparinux
            - selective factor Xa inhibitor
            - indicated in patients receiving initial thrombolytic therapy or with no specific reperfusion therapy
- complications of STEMI
    - pulmonary oedema
        - secondary to left ventricular failure
        - increasing breathlessness, reduced arterial oxygen saturations, tachycardia, third heart sound, pulmonary crepitations
        - exclude other causes of heart failure such as arrhythmias and valvular abnormalities
        - CXR to confirm pulmonary congestion
        - echo to qualify mechanical left ventricular function (e.g. ventricular wall akinesis, impaired ejection fraction)
        - management:
            - treatment of STEMI (i.e. reperfusion)
            - diuretics
            - IV nitrates
            - NIV
            - ACE inhibitors
            - intubation where appropriate
    - cardiogenic shock – left ventricular dysfunction
        - indicates significant myocardial damage
        - usually a consequence of anterior AMI or in the presence of LBBB
        - features:
            - tachycardia
            - reduced systolic blood pressure
            - cool peripheries
            - reduced renal output
        - associated with adequate central venous filling pressures
        - clinical assessment should exclude other causes of hypotension (e.g. hypovolaemia, arrhythmias, drug-related side effects, right ventricular infarction, mechanical complications of STEMI)
        - management:
            - early and effective myocardial reperfusion (PPCI if available)
            - may require inotropes or IABP, particularly where there is evidence of poor cerebral or renal perfusion
    - right ventricular dysfunction
        - presents with hypotension and distended neck veins but without signs of pulmonary oedema
        - should prompt right sided chest lead (V4R) which may show elevation
        - echo
            - demonstrates varying degrees of right ventricular myocardial wall dysfunction and/or functional tricuspid incompetence due to ventricular dilatation

- recognition is important as management is significantly different from management of cardiogenic shock caused by left ventricular dysfunction
- right ventricular preload must be maintained
    - nitrates and opiates reduce preload and can cause significant drop in blood pressure
- fluid challenge maintains venous return – should have careful haemodynamic monitoring
- rapid reperfusion is also required

- mechanical complications of STEMI
    - mitral regurgitation
        - commonest mechanical complication
        - signs of acute cardiac failure develop
        - underlying pathological causes include:
            - chordal rupture
            - papillary muscle infarction and rupture
            - functional regurgitation due to dilatation of the mitral valve ring associated with left ventricular dilatation
        - clinical signs of sudden haemodynamic deterioration with new murmur are suggestive
        - diagnosis is confirmed with echo and distinguishes mitral regurgitation from ventricular septal rupture
        - management
            - initially control pulmonary oedema and support the circulation
            - definitive treatment is urgent surgery and reperfusion of the affected coronary artery
    - ventricular septal rupture
        - occurs most commonly with anterior or posterior AMI
        - clinical presentation is similar to mitral regurgitation: haemodynamic compromise and pan-systolic murmur
            - echo is required to distinguish between the two
        - interim measures are to support circulation and reperfusion of the affected artery
        - definitive treatment is surgery
    - cardiac rupture and tamponade
        - acute cardiac rupture is a cause of sudden death following AMI
            - more common in the elderly
        - in subacute rupture the pericardium contains the blood loss and tamponade develops
            - sudden haemodynamic decompensation with signs of tamponade:
                - hypotension
                - distended neck veins
                - muffled heart sounds
        - pericardiocentesis may temporarily improve the haemodynamic state but urgent surgery is required

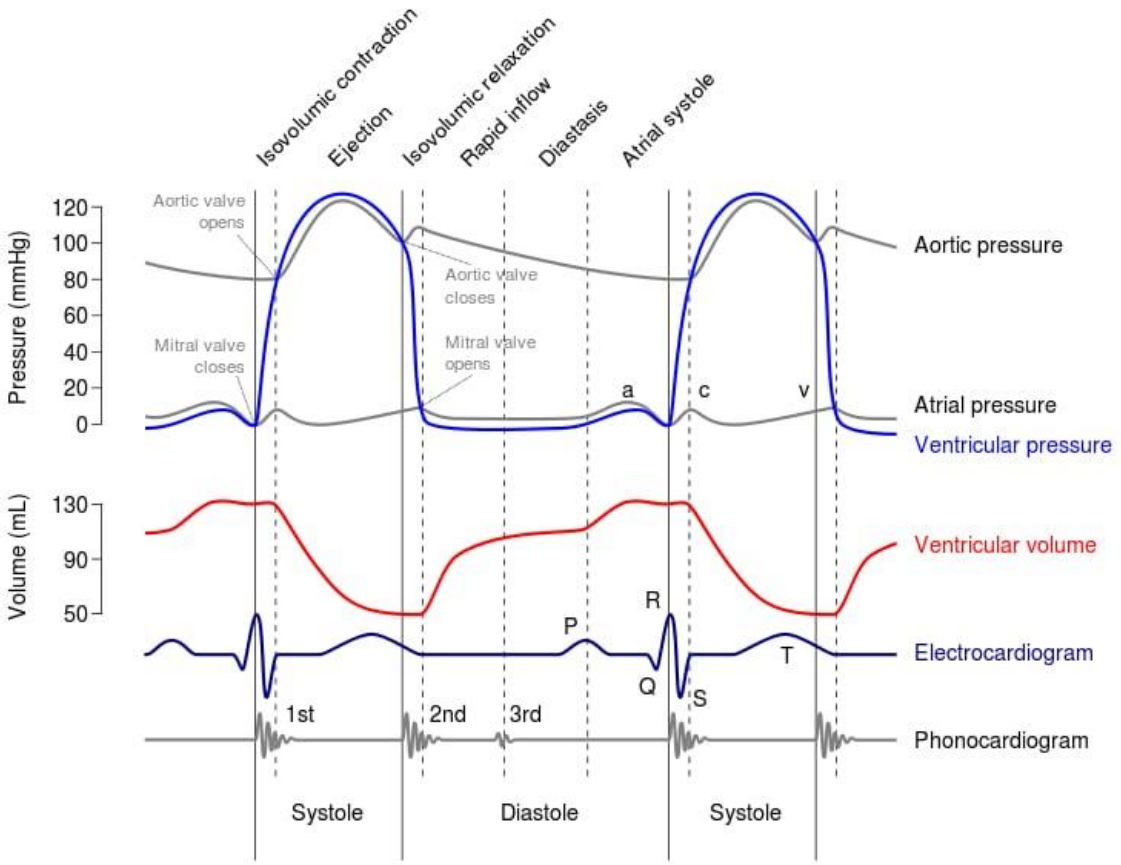
- NSTEMI
    - specific therapies for NSTEMI and unstable angina
        - aspirin
            - commenced at presentation and continued daily
        - clopidogrel
            - 300mg immediately followed by 75mg daily
            - NICE guidance recommends continuation for 12 months
        - ticagrelor
            - may be used as an alternative to clopidogrel in some places
        - LMWH
            - enoxaparin normally used
        - fonadaparinux
            - indicated for NSTEMI patients in whom urgent (<120 minutes) PCI is not indicated
            - NICE recommend it over heparin in the non-urgent setting due to more favourable efficacy/safety profile
        - GpIIb/IIIa inhibitors
            - NICE recommend tirofiban or eptifibatide in patients with NSTEMI who are at higher risk even when early PCI is not planned
        - nitrates
            - provide symptomatic benefit due to systemic and coronary vasodilatory effects
            - patients with NSTEMI who require admission may be started on IV nitrates in the absence of contraindications
                - commonly used for patients with ongoing ischaemic chest pain
        - beta blockers
            - reduce myocardial oxygen demand by reducing cardiac contractility and cardiac rate through their effects on beta-adrenergic receptors in the myocardium, sino-atrial node and atrio-ventricular node
                - inhibit the effects of circulating catecholamines
            - should be used in patients with NSTEMI in the absence of contraindications (e.g. acute left ventricular failure, hypotension, bradycardia)
            - IV or oral routes are acceptable
            - most commonly used are atenolol, metoprolol or bisoprolol
        - calcium channel blockers
            - three physiological effects:
                - vasodilation
                - reduced myocardial contractility
                - reduced heart rate (via delayed atrio-ventricular conduction)
    - summary of NICE recommendations for initial management of non-ST elevation ACS
        - GRACE score (6 month mortality) 1.5% or below (lowest risk)
            - admit to medical team
            - cardiology review within 24 hours
            - aspirin

        - 300mg stat then 75mg daily
    - bisoprolol
        - 2.5-5mg daily
    - fondaparinux
        - 2.5mg SC daily (unfractionated heparin if eGFR <20)
- GRACE score (6 month mortality) 1.5-3%
    - admit to medical team
    - cardiology review within 24 hours
    - aspirin
        - 300mg stat then 75mg daily
    - bisoprolol
        - 2.5-5mg daily
    - fondaparinux
        - 2.5mg SC daily (unfractionated heparin if eGFR <20)
    - clopidogrel
        - 600mg stat then 75mg daily
- GRACE score (6 month mortality) >3%
    - admit to CCU or cardiology ward
    - aspirin
        - 300mg stat then 75mg daily
    - bisoprolol
        - 2.5-5mg daily
    - fondaparinux
        - 2.5mg SC daily (unfractionated heparin if eGFR <20)
    - clopidogrel
        - 600mg stat then 75mg daily
    - tirofiban
        - infusion as per protocol continued for 72 hours
    - early angiography
        - within 96 hours of presentation
    - isosorbide dinitrate
        - infusion as per protocol only if ongoing pain

CC3 arrhythmias

- initial approach
    - fast or slow?
    - ventricular or supraventricular?
    - compromised or not?
    - does it require management?
    - what is the underlying substrate predisposition?
    - what is the trigger?
    - will it recur?
- bradycardia
    - blocks and bradycardia usually caused by impaired automaticity or conduction
    - if one pacemaker fails, another generally takes over at a lower rate
- tachycardia
    - re-entrancy

        - the impulse reaches a point where it can go two ways – if one is blocked it must go the other way
            - when the impulse reaches a point where the two pathways rejoin it may be retrogradely conducted up the blocked pathway until it reaches the beginning and travels down the path creating a re-entry loop
        - the block that leads to re-entry is often transient and timing-dependent
        - sometimes they do not occupy a fixed anatomical location
    - automaticity (increased/abnormal)
        - increased
            - normal spontaneous depolarisation is increased for some reason (adrenergic stimulation)
        - abnormal
            - local ischaemia
            - hypokalaemia
            - drugs
    - triggered activity
        - 'after depolarisation' – the normal action potential suddenly swings positive again allowing another depolarisation to occur abnormally
        - early after depolarisations – occur before depolarisation has finished
        - delayed after depolarisations – occur after membrane potential has returned to normal (from raised intracellular calcium)
- factors contributing to arrhythmogenesis
    - structural
        - MI
        - hypertrophy
        - myopathic ventricle
        - ischaemia
        - congenital defects
    - systemic
        - hypoxia
        - acidosis
        - electrolytes
        - catecholamines
    - drugs
        - catecholamines
        - proarrhythmic drugs
        - anti-arrhythmic drugs in certain situations
            - toxicity (increased dose or reduced clearance)
            - severe LVF with digoxin
            - bradycardia
- Vaughan Williams classification of antiarrhythmic drugs



https://litfl.com/cardiovascular-physiology-overview/

- class I – inhibit fast sodium channels
  - inhibit fast voltage sensitive sodium channels during depolarisation (phase 0) of the cardiac action potential
    - decreased depolarisation and conduction velocity
    - membrane stabilisers
  - Ia prolonged AP duration
    - decreased rate of phase 0 depolarisation
      - reduces excitability of non-nodal regions in the heart important for propagation of the action potential
    - lengthen the duration of the action potential
    - e.g. quinidine, procainamide
  - Ib – shortened AP duration
    - decreases the rate of spontaneous phase 4 outside the atria
      - decreases automaticity
    - e.g. lignocaine, phenytoin
  - Ic – no change in AP duration
    - potent sodium channel blockers
      - decrease in rate of phase 0 depolarisation and speed of conduction of cardiac impulses
    - little effect on the duration of the cardiac action potential and effective refractory period in ventricular myocardial cells

                - shortens the duration of the action potential in Purkinje fibres
                - e.g. flecainide
        - class II – decreased rate of depolarisation (beta blockers)
            - beta-adrenoceptor antagonists
            - increase effective refractory period of AV node and decrease automaticity, decrease QT duration
                - decreased heart rate and O2 consumption
            - e.g. metoprolol, esmolol, propranolol
        - class III – inhibit potassium ion channels
            - prolong the refractory period by blocking potassium channels
                - prolonged cardiac depolarisation, action potential duration and effective refractory period
                - decreased time in which the cardiac muscle cells are excitable
            - e.g. amiodarone, sotalol
                - amiodarone has some class I, II, III and IV effects
        - class IV – inhibit slow calcium channels
            - inhibit inward slow calcium ion currents that may contribute to development of VT
                - impair SA node pacemaker activity
            - decreased duration of action potential but no effect on automaticity
            - e.g. verapamil, diltiazem, nifedipine
- life-threatening arrhythmias
    - tachycardia
        - adverse features
            - shock
            - syncope
            - myocardial ischaemia
            - heart failure
        - if adverse features, proceed to synchronised DC shock, up to 3 attempts
            - 120-150J biphasic increased in increments for broad complex tachycardia or atrial fibrillation
            - 70-120J biphasic increased in increments for atrial flutter or regular narrow complex tachycardia
            - if unsuccessful, amiodarone 300mg IV over 10-20 minutes followed by repeat shock
                - then amiodarone 900mg over 24 hours
        - if no adverse features
            - broad complex (>0.12s)
                - regular
                    - VT or uncertain
                        - amiodarone 300mg IV over 20-60 minutes then 900mg over 24 hours
                    - SVT with bundle branch block
                        - treat as narrow complex tachycardia
                - irregular
                    - expert help should be sought

- AF with bundle branch block – treat as for narrow complex
- pre-excited AF – consider amiodarone

- narrow complex (<0.12s)
  - regular
    - vagal manoeuvres
    - adenosine 6mg, 12mg, 18mg
    - continuous ECG recording
    - if sinus rhythm achieved, probably re-entry paroxysmal SVT
      - record 12 lead in sinus rhythm
      - if recurs, treat again and consider anti-arrhythmic prophylaxis
    - if sinus rhythm not achieved, seek expert help
      - probable atrial flutter
        - control rate (e.g. beta-blocker)
  - irregular
    - probable AF
      - control rate with beta-blocker or diltiazem
      - if in heart failure consider digoxin or amiodarone
      - assess thromboembolic risk and consider anticoagulation

- bradycardia
  - adverse features
    - shock
    - syncope
    - myocardial ischaemia
    - heart failure
  - if adverse features:
    - atropine 500mcg IV
      - if no satisfactory response, consider interim measures:
        - repeat atropine 500mcg IV up to maximum of 3mg
        - transcutaneous pacing
        - isoprenaline 5mcg/min IV
        - adrenaline 2-10mcg/min IV
        - alternative drugs:
          - aminophylline
          - dopamine
          - glucagon (if bradycardia caused by beta blocker or calcium channel blocker)
          - glycopyrrolate (may be used instead of atropine)
        - arrange transvenous pacing
  - if no adverse features:
    - no risk of asystole
      - continue observation

- risk of asystole
    - recent asystole
    - Mobitz II AV block
    - complete heart block with broad QRS
    - ventricular pause >3s
  - consider interim measures as above

- atrial fibrillation
  - assessment of stroke and bleeding risk
    - CHA_2DS_2-VASc score for stroke risk for anyone with:
      - symptomatic or asymptomatic paroxysmal, persistent or permanent atrial fibrillation
      - atrial flutter
      - continuing risk of arrhythmia recurrence after cardioversion back to sinus rhythm or catheter ablation
    - CHA_2DS_2-VASc score
      - age
        - <65 (0)
        - 65-74 (1)
        - ≥75 (2)
      - sex
        - female (1)
        - male (0)
      - CHF history
        - no (0)
        - yes (1)
      - hypertension history
        - no (0)
        - yes (1)
      - stroke/TIA/thromboembolism history
        - no (0)
        - yes (2)
      - vascular disease history (prior MI, peripheral artery disease, aortic plaque)
        - no (0)
        - yes (1)
      - diabetes history
        - no (0)
        - yes (1)
      - 0 = low risk (may not require anticoagulation)
      - 1 = low-moderate risk (consider antiplatelet or anticoagulation)
      - 2 or more = moderate-high risk (anticoagulation recommended)
  - bleeding risk
    - should be assessed when:
      - considering starting anticoagulation in people with atrial fibrillation
      - when reviewing people already taking anticoagulation
    - ORBIT bleeding risk
      - **o**lder age (≥75) (1 point)

- **r**educed haemoglobin/haematocrit (2 points)
    - Hb <130 in males, <120 in females
    - hct <40% in males, <30% in females
    - history of anaemia
- **b**leeding history (2 points)
- **i**nsufficient renal function (1 point)
    - eGFR <60
- **t**reatment with antiplatelet agents (1 point)
- 0-2 – low risk
- 3 – medium risk
- 4-7 – high risk

- results of stroke and bleeding risks should be discussed with the patient, taking into account their co-morbidities and their preferences
- information for patients (generally from GP or medics/cardiologists) with atrial fibrillation should be personalised and include:
    - stroke awareness and measures to prevent stroke
    - rate control
    - assessment of symptoms for rhythm control
    - who to contact for advice if needed
    - psychological support if needed
    - up to date and comprehensive education and information on:
        - cause, effects and possible complications of atrial fibrillation
        - management of rate and rhythm control
        - anticoagulation
        - support networks
- anticoagulation
    - should be offered to anyone with a CHA_2DS_2-VASc score of 2 or above (or men with a score of 1 or above), taking into account bleeding risk
    - apixaban, dabigatran, edoxaban and rivaroxaban are recommended as options
    - consider vitamin K antagonist if DOAC contraindicated, not suitable or not tolerated
    - anticoagulation should not be offered to people with atrial fibrillation under 65 with no other risk factors other than their sex
    - anticoagulation should not be withheld solely due to age or falls risk
- rate control
    - should be offered as a first-line treatment strategy for atrial fibrillation except in people:
        - whose atrial fibrillation has a reversible cause
        - who have heart failure thought to be primarily caused by atrial fibrillation
        - with new onset atrial fibrillation
        - with atrial flutter thought to be appropriate for ablation to restore sinus rhythm
        - for whom a rhythm control strategy would be more appropriate based on clinical judgement

                - standard beta-blocker (i.e. not sotalol) or rate-limiting calcium channel blocker (diltiazem or verapamil)
                - digoxin can be considered if the person does little or no exercise or other rate-limiting drug options are ruled out
        - rhythm control
            - consider if symptoms continue after rate has been controlled or rate control strategy not successful
            - standard beta-blocker is first line option
            - amiodarone can be considered for people with heart failure or left ventricular impairment
            - patients with paroxysmal atrial fibrillation may have 'pill in pocket' management
- NICE guidance for acute presentation of atrial fibrillation
    - emergency electrical cardioversion if life-threatening haemodynamic instability
    - otherwise:
        - either rate or rhythm control if onset less than 48 hours
        - rate control if onset more than 48 hours or uncertain
    - if there is decompensated heart failure, beta blockers require specialist input and calcium-channel blockers must be avoided
    - pharmacological cardioversion
        - flecainide or amiodarone for people with no evidence of structural or ischaemic heart disease
        - amiodarone if evidence of structural heart disease
    - if duration >48 hours or uncertain, cardioversion should be delayed until therapeutically anticoagulated for at least 3 weeks with rate control in the meantime
    - for new onset AF, heparin should be given until appropriate antithrombotic therapy started

CC4 cardiac failure

- severe cardiac failure
    - overview
        - preload reduction
            - diuretics, opioids, decreased intake, spironolactone
        - afterload reduction
            - ACEi, GTN, IABP
        - increased contractility
            - milrinone, dobutamine, adrenaline, VAD
        - decreased myocardial work
            - beta-blockers, IABP, VAD
        - increased coronary perfusion and oxygenation
            - O2, Hb, stents, CABG, IABP
    - acute medical management
        - sit patient up
        - high flow oxygen
        - nitrates (IV, sublingual, topical) if not hypotensive
        - NIV +/- intubation

                - CPAP
                    - favourable effects on intrathoracic and left ventricular transmural pressure
                    - significant reduction in mortality and intubation rates
                - BIPAP
                    - reduction in intubation, trend to reduction in mortality rates
                - invasive ventilation
                    - poor prognosis but can produce dramatic improvement
            - urinary catheter
            - DVT prophylaxis
            - inotropes
                - used as bridge to transplant or revascularisation
            - diuretics if evidence of fluid overload
                - relieves symptoms, no survival advantage
            - avoid opioids
                - higher rates of intubation
            - depending on patient, IABP, VAD or ECMO may be considered
                - particularly if transient cause or as bridge to transplant
    - chronic management
        - loop diuretics
            - symptomatic relief, no mortality benefit
        - ACEi and ARBs
            - improved mortality and hospital admission
        - beta-blockers
            - mortality reduction
        - aldosterone inhibitors – spironolactone
            - marked mortality reduction
        - anticoagulation
            - for very low ejection fraction or AF
        - digoxin
            - no mortality advantage
        - biventricular pacing
            - may benefit some patients
        - AF
            - rate control with digoxin, amiodarone and beta-blockers plus anticoagulation
        - ventricular arrhythmias
            - may require ICD
    - invasive interventions
        - may require urgent surgery for acute MR or AR with acute pulmonary oedema
        - LVAD – may be used as bridge to transplant or for those not eligible for transplant
            - mortality reduction but major complications
        - revascularisation
            - patients with cardiogenic shock do better with revascularisation
- drugs to avoid in heart failure

    - pro-anti-arrhythmics with potentially negative inotropic effects (e.g. flecainide)
    - calcium channel blockers (e.g. verapamil, diltiazem)
        - only amlodipine is advisable
    - tricyclic antidepressants
    - lithium
    - NSAIDs and COX-2 inhibitors
    - corticosteroids
    - drugs prolonging QT interval and potentially precipitating ventricular arrhythmias (e.g. erythromycin, terfenadine)
- aetiology
    - valvular heart disease
        - aortic stenosis
            - can cause LVH due to chronic excessive afterload
        - AR, MR, ASD, VSD and tricuspid incompetence cause excessive preload
    - myocardial disease
        - coronary heart disease
        - hypertension
            - increased vascular resistance, often with LVH but preserved ejection fraction
        - cardiomyopathies
        - drugs
            - beta blockers
            - calcium antagonists
            - anti-arrhythmics
            - cytotoxics
        - toxins
            - alcohol
            - cocaine
            - mercury
            - cobalt
            - arsenic
        - endocrine
            - diabetes
            - hypothyroidism
            - hyperthyroidism
            - Cushing's syndrome
            - adrenal insufficiency
            - excessive growth hormone
            - phaeochromocytoma
        - nutritional
            - obesity
            - cachexia
            - deficiency of:
                - thiamine
                - selenium
                - carnitine
        - infiltrative

                - sarcoidosis
                - amyloidosis
                - haemochromatosis
                - Löffler's eosinophilia
                - connective tissue disease
            - infective
                - Chagas' disease
                - HIV
    - arrhythmias
    - high output cardiac failure (where there is increased peripheral demand on the heart)
        - anaemia
        - pregnancy
        - sepsis
        - hyperthyroidism
        - Paget's disease of the bone
        - arteriovenous malformations
        - beriberi
- symptoms
    - dyspnoea on exertion and fatigue
    - orthopnoea
    - paroxysmal nocturnal dyspnoea
    - fluid retention
        - may cause pulmonary or peripheral oedema
            - with peripheral oedema the patient may complain of weight gain, ankle swelling or bloating
    - nocturnal cough (+/- pink frothy sputum) or wheeze
    - anorexia
- signs
    - tachycardia
    - tachypnoea
    - pulmonary rales
    - pleural effusion
    - raised JVP
    - peripheral oedema
    - hepatomegaly
- investigations for heart failure
    - ECG
    - CXR
        - cardiomegaly
        - ventricular hypertrophy
        - prominent upper lobe veins (upper lobe diversion)
        - peribronchial cuffing
        - diffuse interstitial or alveolar shadowing
            - perihilar 'bat's wings' or nodular
        - fluid in the fissures
        - pleural effusions

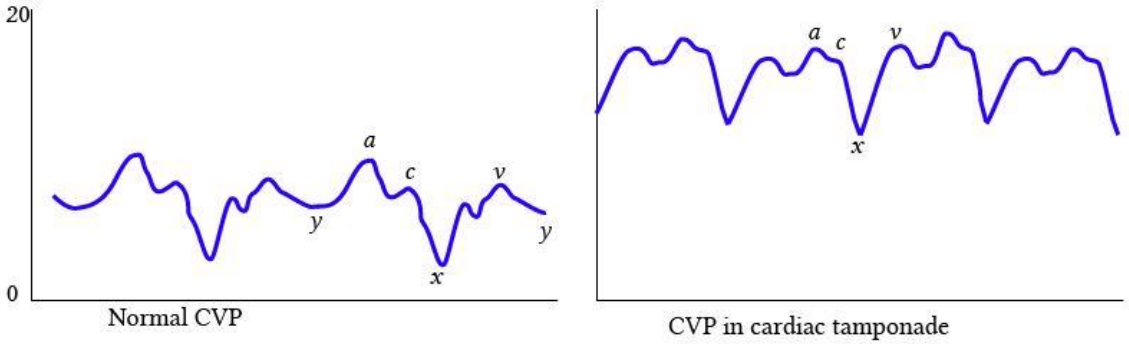
        - Kerley B lines
    - bloods
        - U&E
        - FBC
        - TFTs
        - LFTs
        - lipid profile
        - HbA1c
        - NT-proBNP
            - also raised in:
                - LVH
                - ischaemia
                - tachycardia
                - hypoxaemia
                - renal dysfunction (eGFR <60)
                - age >70
                - liver cirrhosis
                - sepsis
                - COPD
                - diabetes
            - reduced in:
                - obesity
                - African-Caribbean origin
                - medications (diuretics, ACEi, ARBs, beta-blockers, mineralocorticoid receptor antagonists)
    - urinalysis
    - peak flow or spirometry
- New York Heart Association (NYHA) classification of heart failure:
    - class I
        - no symptoms on ordinary physical activity
    - class II
        - slight limitation of physical activity by symptoms
    - class III
        - less than ordinary activity leads to symptoms
    - class IV
        - inability to carry out any activity without symptoms
- prognosis
    - poor – around 50% die within 5 years of diagnosis
    - generally, the lower the ejection fraction, the poorer the prognosis
    - other poor prognostic factors include:
        - increasing age
        - smoking
        - diabetes and other co-morbidities
        - obesity or low BMI

CC5 cardiac tamponade

- causes of pericardial effusion

    - vascular
        - aortic root rupture
        - aortic dissection (Type A)
        - myocardial infarction
    - infective/inflammatory
        - pericarditis of any sort
    - neoplastic
        - malignant infiltration of the pericardial sac
        - post-radiotherapy
    - drug-induced
        - isoniazid
        - cyclosporine
        - hydralazine
    - idiopathic
    - congenital
        - congenital hypothyroidism
    - autoimmune
        - SLE
        - RA
        - CREST
        - sarcoidosis
    - traumatic
        - injury (penetrating)
        - surgery (e.g. CABG)
    - endocrine
        - hypothyroidism
- mechanism
    - pericardium is an inextensible fibrous sac
    - small volumes quickly give rise to a rapid increase in pericardial pressure and tamponade physiology
        - this produces decreased cardiac output because the stroke volume is limited by the presence of incompressible pericardial fluid
- clinical features
    - nonspecific findings
        - tachycardia
        - tachypnoea
    - Beck's triad
        - muffled heart sounds
        - hypotension
        - raised CVP
    - Kussmaul's sign
        - neck veins distend with inspiration instead of collapsing
    - Friedrich's sign
        - an exaggerated early drop in diastolic CVP
        - rapid y descent followed by rapid rise in pressure
    - pulsus paradoxus
        - exaggeration of the normal inspiratory fall in blood pressure

        - leads to radial pulse not being palpable during inspiration whilst heart sounds can be auscultated
        - can be clinically elicited by:
            - 'swing' on invasive arterial pressure trace
            - palpation of the radial pulse disappearing on inspiration
            - sphygmomanometry – with blood pressure cuff elevated, Korotkoff sounds disappear during inspiration
            - pulse oximetry – waveform changes in the same way as the arterial trace
    - pericardial rub
        - if tamponade associated with inflammation
    - pericardial knock
        - probably the result of a sudden cessation in ventricular filling
    - third heart sound
        - may be confluent with or the same as the pericardial knock
    - displaced apex beat
        - not mandatory but can happen with chronic effusions
    - characteristic CVP findings
        - sawtooth M or W configuration of raised CVP
        - CVP is raised
        - all CVP elements are raised
        - a and v waves are tall
        - x descent is steep
        - y descent is (usually) absent
            - because early diastolic blood flow from the right atrium to the right ventricle is impaired by the compressive effect of the surrounding pericardial fluid



https://derangedphysiology.com/main/required-reading/cardiac-arrest-and-resuscitation/Chapter%20221/cardiac-tamponade

- ECG findings in tamponade
    - tachycardia
    - low QRS voltage trace
        - large amount of fluid with poor conductivity between the heart and the electrodes

    - electrical alternans
        - alternating high and low QRS complexes
        - usually associated with massive pericardial effusion
        - due to the contracting heart changing position rhythmically within the pericardial sac
    - global concave ST elevation
        - injury from direct pressure on the myocardium
    - PR depression
        - usually associated with pericarditis
    - T wave inversion
        - may develop but not unique to tamponade
- echocardiogram
    - visible pericardial effusion
        - may not be seen with blood clot rather than fluid
    - diastolic collapse of right atrium and right ventricle
        - when chamber pressures are lower than pericardial fluid pressure
    - right atrial collapse in systole
        - atrial cavity pressure is lower than the pericardial fluid pressure in early systole
        - collapse for longer than one third of the cardiac cycle is 100% specific for clinical cardiac tamponade
    - right ventricular collapse in diastole
        - only present in expiration initially
        - the longer the duration of the collapse, the more severe the tamponade
    - septal bounce
        - inspiratory movement of the septum towards the LV
    - IVC dilatation
        - because all veins are dilated – echo equivalent of raised JVP
- management
    - 2 large bore cannulae
    - supplementary oxygen
    - IV fluid bolus
    - pericardiocentesis
        - equipment
            - 18g spinal needle or large bore needle from central line kit
            - 20ml syringe
            - sterile field and sterile gown/gloves
        - blind approach
            - if US not available
            - pericardium is 2-3cm below skin in average adult
            - use 16-18g needle attached to large syringe
            - chest wall should be cleaned and draped
            - insert needle at the xiphoid process up into the chest at a 45 degree angle, directed towards the tip of the left scapula
            - aspirate syringe as it advances every 1-2mm until fluid is aspirated
            - it should be possible to feel the needle penetrate the pericardium

        - if immediate surgical management (thoracotomy) not available, a catheter can be advanced using the Seldinger technique to allow for continuous drainage
- US-guided approach
    - best views are based on body habitus, positioning and which axis of the heart is optimally viewed
        - subxiphoid or parasternal long axis views are most commonly used
    - best probe is low frequency phased array but a curvilinear one can be used
    - subxiphoid approach
        - probe placed in subxiphoid area angled up into the chest using the liver as a window
        - pericardial effusion seen as black anechoic area above the right ventricle which should be where the needle enters
        - distance can be measured from where the needle will be inserted to the pericardial effusion
        - needle should be inserted parallel to the probe and directed at 45 degree angle towards the tip of the left scapula
        - syringe should be aspirated every 1-2mm until fluid is drawn back
    - parasternal approach
        - probe placed on chest wall in left parasternal position at around 4^{th} intercostal space
        - needle should be inserted at a 45 degree angle in-plane to the probe on the anterior chest wall and directed down towards the effusion
        - the ideal insertion site should be closest to where the effusion is largest
- confirmation
    - agitated saline flushed through the syringe into the pericardial space can create a snowstorm appearance on US
    - fluid or non-clotted blood will be aspirated
    - removal of 5-10ml of fluid can increase stroke volume by 25-50% and result in a dramatic increase in cardiac output and blood pressure
- complications
    - dry tap
    - pneumothorax
    - myocardial injury
    - arrythmias
    - cardiac arrest
        - needle can perforate the RV or a coronary artery resulting in a worsening tamponade
    - liver injury with subxiphoid approach

CC6 congenital heart disease

- background
    - most common congenital malformation
    - affects 9 in 1000 newborns
- pathophysiology
    - foetal circulation
        - foetal circulation has three shunts to direct blood away from the developing lungs and liver
        - lungs are filled with fluid, causing a high resistance to pulmonary blood flow
        - oxygen and nutrient rich blood comes through the placental vein, through the ductus venosus into the foetal IVC, bypassing the liver
        - the blood reaches the right atrium and is shunted to the left atrium via the foramen ovale
            - the foramen ovale acts a flap valve and is held open by the difference in pressure between the right and left atria
                - the left atrium has a low pressure because there is little blood returning from the lungs, whereas pressure in the right atrium is higher as it receives systemic venous return
            - a small amount of blood passes into the right ventricle and pulmonary trunk and is shunted to the aorta via the ductus arteriosus to bypass the non-functioning lungs
        - blood from the left atrium passes through to the left ventricle and is pumped around the foetus via the aorta
        - de-oxygenated blood flows back to the placenta via the umbilical arteries
    - duct dependent lesions
        - timing of presentation relates to whether lesions are duct-dependent or not
            - neonates with duct dependent lesions are dependent on the ductus arteriosus remaining open to allow mixing of blood to maintain systemic or pulmonary circulation
        - presentation is usually around the time the DA closes
            - this happens due to withdrawal of prostaglandin E2
                - levels fall at birth due to proper functioning of the lungs, changes in neonatal blood pressures in the systemic and pulmonary circulations and removal of the placenta
- shock and the collapsed baby
    - shock results from inadequate oxygen delivery to meet metabolic demands of the tissues
    - five main causes of collapse in the neonate are:
        - sepsis
        - congenital heart disease
        - metabolic/endocrine disturbances (including inborn errors of metabolism)
        - NAI
        - trauma
- mnemonic for common causes of neonatal collapse (THEMISFITS)
    - **t**rauma
    - **h**eart disease
    - **e**ndocrine

    - **m**etabolic/electrolyte disturbance
    - **i**nborn errors of metabolism
    - **s**eizures/CNS abnormalities
    - **f**ormula errors (e.g. overdilution)
    - **i**ntestinal disorders (e.g. intussusception)
    - **t**oxins
    - **s**epsis
- congenital heart disease lesions presenting as shock
    - coarctation of the aorta (severe)
        - significant narrowing of thoracic aorta just distal to the left subclavian artery near the region of the ductus arteriosus
        - can be pre-ductal, ductal or post-ductal
        - vessels to the head and upper limbs emerge proximal to the narrowing so blood supply to these areas is not compromised
        - blood flow to the rest of the body is often reduced
            - can cause radio-femoral delay before the DA closes or absent femoral pulses after
        - co-arctation causes increased afterload on the left ventricle which can cause hypertrophy
        - coarctation is not always severe
            - lesions are critical when the systemic circulation depends on ductus arteriosus patency
            - can remain asymptomatic until adulthood
            - often associated with other abnormalities such as VSD or bicuspid aortic valve
        - examination
            - absent femoral pulses or radio-femoral delay before DA closes
            - cyanosis
            - 4 limb BP discrepancies
                - gradient of >10mmHg between upper and lower limbs is clinically significant
            - signs of CCF
        - investigations
            - diagnosis usually made prenatally on US
            - ECG may show LVH or RV conduction delay
            - CXR may show figure of 3 sign

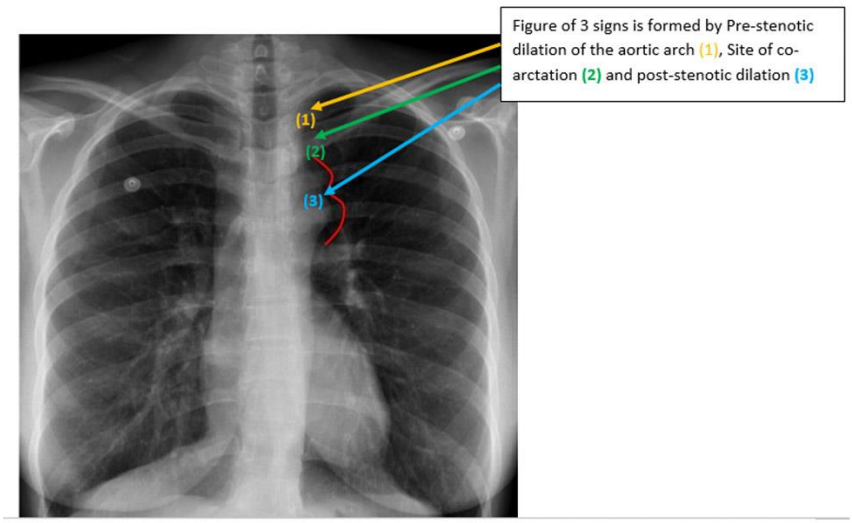


Figure of 3 signs is formed by Pre-stenotic dilation of the aortic arch (1), Site of co-arctation (2) and post-stenotic dilation (3)
(1)
(2)
(3)

https://www.rcemlearning.co.uk/reference/congenital-heart-disease-2/#1605865843029-ec84a4ea-76a8

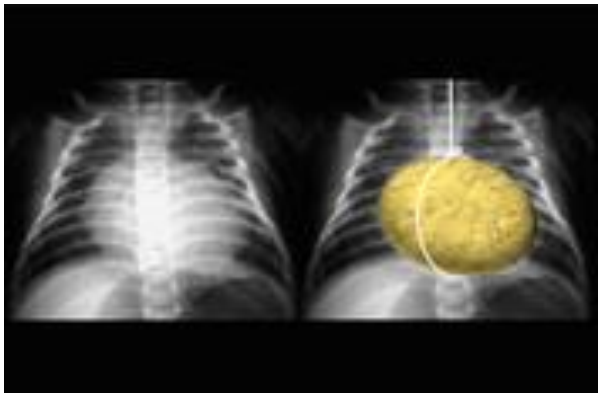
- patients will have cardiac MRI or CT for evaluation of thoracic aorta prior to management

- management
  - rapid A-E assessment
  - prostaglandin infusion if duct-dependent circulation
  - diuretics to reduce preload combined with volume replacement to correct metabolic acidosis
  - surgery usually performed within 24 hours and removes the narrow segment

- hypoplastic left heart syndrome
  - lesion causes severe mitral and aortic valve stenosis or atresia
    - associated with an underdeveloped left ventricle, ascending aorta and aortic arch – often too small to maintain cardiac output
    - an ASD is also necessary to allow the pulmonary venous blood flow from the left atrium to the right atrium
    - a PDA is necessary to allow blood flow from the pulmonary artery to the systemic circulation – shock occurs when it closes
  - it is a common diagnosis (around 260/year in the UK)
  - usually diagnosed prenatally
  - most patients are initially stable
    - over several days, as pulmonary vascular resistance falls and the PDA begins to close, the ratio of pulmonary to systemic blood flow will increase, causing increased right to left shunting
      - this leads to cyanosis, congestive heart failure, shock and respiratory distress
  - examination
    - absent femoral and brachial pulses, delayed capillary refill time, hypotension secondary to shock
    - signs of right heart failure – enlarged liver, tachypnoea, rales
    - shock and respiratory distress secondary to acidosis on DA closure
    - may have PDA murmur

        - management
            - patients should be born in a cardiac centre
            - prostaglandin infusion to maintain DA patency
            - surgery within 3-5 days
                - staged management with 3 operations to establish a single ventricle circulation
                    - Norwood procedure (neonatal period)
                        - creation of common atrium by removing atrial septum
                        - pulmonary arteries attached to a reconstructed aortic arch to create a single outlet right sided heart
                        - pulmonary circulation provided by BT shunt or an RV to PA conduit
                    - Glenn procedure (4-6 months)
                        - SVC connected to pulmonary arteries
                        - venous return from the head and neck is routed directly to the lungs which allows saturations to reach 80-85%
                        - the BT shunt/conduit is removed
                        - by 3-5 years there will be insufficient blood returning from the head and neck to keep the child well
                    - Fontan procedure (3-5 years)
                        - IVC connected to the pulmonary circulation so venous return from the body is routed directly to the lungs which allows saturations to reach 90-95%
                        - most patients with a Fontan circulation reach adulthood but must have long term follow up
                        - many can manage school/jobs and some give birth
- cyanosis in the neonate
    - background
        - can be central or peripheral
            - central is always pathological, peripheral may not be
            - in the context of CHD it is caused by right to left shunting
                - deoxygenated blood mixes with oxygenated blood and is pumped round the body
        - cyanosis can be harder to detect in dark skinned or anaemic patients so must be carefully looked for
    - main CHD lesions that cause cyanosis
- truncus arteriosus
    - 1 common artery
- transposition of the great arteries
    - 2 switched arteries

- tricuspid atresia
    - 3 leaflets of the tricuspid valve are underdeveloped
- tetralogy of Fallot
    - 4 defects with one cause
- TAPVD
    - 5 words make up the lesion name
    - truncus arteriosus
        - pathophysiology
            - the only artery arising from the heart is the common truncus arteriosus
                - functions as both the aorta and pulmonary artery
            - cyanosis occurs due to mixing of oxygenated and deoxygenated blood
            - there is a single semilunar valve
            - lesion is often associated with a VSD which sits under the semilunar valve
            - as pulmonary resistance falls after birth, more and more blood travels into the low resistance pulmonary circuit and heart failure develops
        - examination
            - wide pulse pressure
            - systolic ejection murmur – increased flow across the semilunar valve
            - single second heart sound
        - management
            - surgical repair via a Rastelli procedure
                - involves a patch to close the VSD and allow the LV to communicate with the common trunk
                - a conduit is then made from the RV to the pulmonary artery
    - transposition of the great arteries
        - pathophysiology
            - occurs due to failure of septation of the pulmonary artery and aorta
            - the aorta is connected to the right ventricle and the pulmonary artery is connected to the left ventricle
            - there are two parallel circulations, where deoxygenated blood is pumped around the body and oxygenated blood circles around the lungs
                - unless there is mixing of the parallel circulations (VSD, ASD, PDA), severe cyanosis, metabolic acidosis and death occurs
        - investigations
            - ECG – usually normal
            - CXR – may show ‘egg on string’ sign or signs of CCF



https://radiopaedia.org/articles/egg-on-a-string-sign-heart?lang=us

- management
  - when there is no VSD, cyanosis will occur when the DA closes, requiring prostaglandin infusion
  - if there is a VSD, patients are usually well oxygenated but are more prone to developing CCF so require surgical repair within the first few weeks
  - some infants with TGA and an intact ventricular septum remain excessively cyanotic despite PGE infusion
    - they require balloon atrial septostomy to create an ASD
  - definitive management is an arterial switch procedure, often carried out in the first few weeks to avoid left ventricular deconditioning

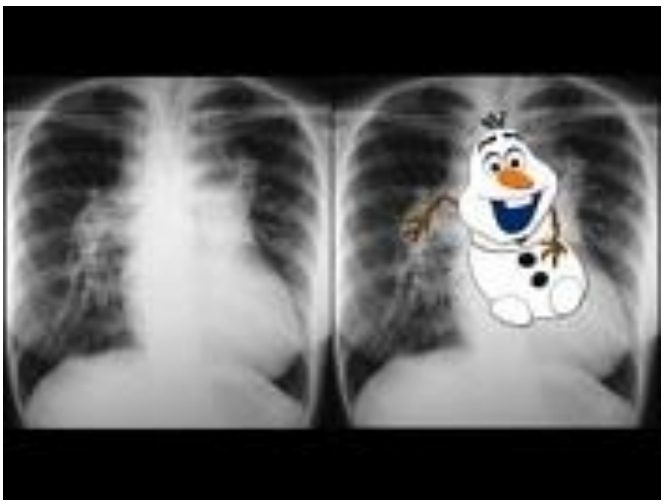
- tricuspid atresia
  - pathophysiology
    - lack of development of the tricuspid valve leading to no inlet to the RV
    - the resulting right ventricle is small and underdeveloped and there is often a degree of pulmonary stenosis
    - causes right to left shunting as systemic venous return enters the right atrium, is shunted through the foramen ovale into the left atrium and left ventricle and into the aorta
    - a variable amount of systemic return gains access to the pulmonary artery via an VSD
      - flow to the pulmonary artery is limited by the size of the ventricular defect and the amount of pulmonary stenosis
      - if there is a large VSD and decreased pulmonary stenosis there is minimal cyanosis at birth but CCF and pulmonary hypertension develop due to increased pulmonary flow
      - if the VSD is small and there is increased pulmonary stenosis, flow into the pulmonary circulation depends on a PDA, BT shunt or RV-PA conduit
  - examination
    - harsh systolic murmur
  - investigations
    - ECG – superior axis, absent RV voltages, large P waves

            - CXR – may have increased or decreased pulmonary vascular markings
        - management
            - if the lesion presents with cyanosis a BT shunt or RV to pulmonary artery conduit is required shortly after birth
            - if it presents with CCF, medical management is used initially followed by surgical correction with a pulmonary artery band to reduce pulmonary artery vascular resistance and pressure
            - definitive management is to establish Fontan circulation by 3-5 years
    - tetralogy of Fallot
        - pathophysiology
            - consists of four lesions occurring together as the result of a single developmental defect
                - VSD
                - overriding aorta
                - pulmonary stenosis
                - resultant RV hypertrophy
            - pulmonary stenosis and RV hypertrophy cause RV pressure to be greater than LV pressure, causing right to left shunting through an often large VSD
            - the overriding aorta sits over the VSD so the mixed blood is pumped to the body
        - presentation
            - depends on degree of RV outflow obstruction which determines the extent of mixing of oxygenated and deoxygenated blood
            - if there is a large degree of pulmonary stenosis the entire right ventricle output passes through the VSD
                - pulmonary flow is only supplied by the DA so they present with cyanosis when the duct closes
            - if there is less pulmonary stenosis, pulmonary blood flow will be higher
                - CCF and cyanosis will occur later in life (hypercyanotic ‘tet spells’)
        - pathophysiology of tet spells
            - occurs when there is hypoxaemia of sudden onset
                - e.g. after systemic vasodilation following a meal or bath
            - hypoxaemia leads to a decrease in systemic vascular resistance, decreased afterload and increased right to left shunting
                - this worsens hypoxaemia, leading to metabolic acidosis and respiratory distress
                - it may progress to unconsciousness or convulsions due to acidosis
        - management of tet spell
            - knees to chest

        - increases preload which may improve flow through pulmonary stenosis and allow more blood to reach the lungs for oxygenation
        - increases afterload, which may allow the left ventricular pressure to prevent as much deoxygenated blood crossing over the VSD into the circulation
    - oxygen
    - morphine
    - get expert help, child is likely to require ICU admission
- investigations
    - ECG
        - RA and RV enlargement
    - CXR
        - boot shaped heart due to RA and RV enlargement resulting in an upturned apex with an absent or diminished pulmonary artery
    - echo/ cardiac MRI +/- cardiac catheterisation – gold standard for diagnosis
- management
    - 10% require BT shunt or RV conduit to pulmonary artery in newborn if severely cyanosed
        - acute cyanosis at birth is managed with prostaglandins
    - most have elective surgical repair at 6-9 months
        - involves a patch to close the VSD and surgical widening of the stenosed pulmonary valve
    - following surgery 85% progress to adulthood

- TAPVD – total anomalous pulmonary venous drainage
    - pathophysiology
        - the four pulmonary veins do not drain into the left atrium but drain into the innominate vein (supracardiac), liver (infracardiac) or coronary sinus/right atrium (cardiac)
            - supracardiac – pulmonary veins drain into the vertical vein which drains into the SVC via the innominate vein
                - causes oxygenated and deoxygenated blood to mix in the right atrium
                - there is usually an ASD which allows shunting of mixed blood to the body
            - infracardiac – pulmonary veins drain into the common pulmonary vein, which drains into the vertical vein; this passes through the diaphragm and drains into the IVC
                - causes oxygenated and deoxygenated blood to mix in the IVC and right atrium
                - mixed blood passes through the VSD to the left heart before being pumped around the body
        - presentation
            - depends on the degree of obstruction between pulmonary veins and right heart

        - in supracardiac and infracardiac lesions, there is usually a higher degree of obstruction which presents at birth with pulmonary oedema, pulmonary hypertension and cyanosis, leading to a collapsed cyanotic neonate
            - degree of obstruction is higher because the pulmonary veins must pass through accessory vessels and structures such as the diaphragm and liver
        - in cardiac lesions there is less obstruction – symptoms of cyanosis and CCF develop within weeks to months
    - investigations
        - ECG – right axis deviation, RVH
        - CXR – snowman sign



https://radiopaedia.org/articles/snowman-sign-total-anomalous-pulmonary-venous-return-1?lang=us

        - definitive imaging - echo, cardiac MRI, angiography
    - management
        - if patients present obstructed
            - emergency surgical correction
        - if non-obstructed
            - medical management of CCF followed by elective surgical repair
- congestive heart failure – breathless baby
    - background
        - caused when the heart is unable to supply blood to meet tissue demands
        - in the context of CHD, heart failure is caused by shunting of blood from left to right
            - oxygenated blood has a higher pressure, so when it is shunted to the right side of the heart there is increased flow through the pulmonary vasculature
            - the congestion results in pulmonary oedema and breathlessness
            - the total volume being pumped into the systemic system is lower, which results in poor tissue perfusion, causing fatigue, poor nutrition and failure to thrive

        - often presents in the first 1-3 months of life as the high pulmonary vascular resistance at birth starts to decline
    - clinical features (may be non-specific)
        - symptoms
            - feeding difficulty
            - sweating/tachypnoea with feeds
            - failure to thrive
            - respiratory distress
            - fussiness
        - signs
            - tachypnoea and laboured breathing
            - rales
            - hepatomegaly
            - cyanosis if severe
            - faltering growth
            - displaced apex beat
            - cool peripheries
            - gallop rhythm
- conditions that may present with CCF
    - ventricular septal defect (VSD)
        - examination
            - usually asymptomatic and undetectable unless large
            - large lesions are defined as being the same size or larger than the aortic valve
                - larger lesions are often associated with pulmonary hypertension and present with CCF after one week
            - may have pansystolic murmur at the lower left sternal edge – usually the louder the murmur, the smaller the hole
        - investigations
            - ECG – biventricular hypertrophy by 2 months
            - CXR - increased pulmonary vascular markings and cardiomegaly
        - management
            - trial of medical management
                - loop diuretics such as furosemide, digoxin, ACEi, beta blockers, spironolactone
                - avoid supplementary oxygen and hyperventilation as they cause pulmonary vascular resistance to fall, which causes increased shunting
            - surgery
                - muscular VSDs often close spontaneously, perimembranous ones do not
                    - these are closed surgically or via percutaneous closure by an interventional cardiologist
                    - surgical intervention is deferred to 3-5 months which improves outcomes
    - atrial septal defect (ASD)
        - pathophysiology

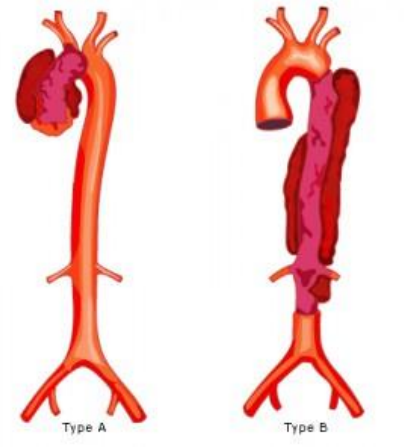
                - openings in the septum between the two atria which persist following birth and can include having a patent foramen ovale
                - causes left to right shunting as pressure in the left atrium is greater than that in the right
                - isolated ASDs rarely cause symptoms in infants as significant shunting does not occur until right ventricular compliance is less than left ventricular compliance
                - leads to CCF at 2-3 years
            - examination
                - soft systolic murmur at upper sternal edge
            - investigation
                - ECG – partial RBBB, RV hypertrophy
                - CXR – cardiomegaly, increased pulmonary vascular markings
            - management
                - medical management to treat symptoms of CCF
                - surgical closure is usually at 3-5 years
                    - 90% have device closure in the catheter lab
                    - 10% have surgical closure of large defects
    - patent ductus arteriosus
        - pathophysiology
            - may exist in isolation of with other types of CHD
            - there is increased pulmonary flow but they are usually asymptomatic in isolation
            - there is left to right shunting so they can cause CCF once the pulmonary vascular resistance falls in the first few months of life
        - examination
            - continuous systolic murmur in the left infraclavicular area
                - known as a machinery murmur
        - investigations
            - ECG – usually normal
            - CXR – increased pulmonary vascular markings if large
        - management
            - 2/3 of PDAs close with a course of indomethacin or ibuprofen (prostaglandin inhibitors)
            - if large enough to cause CCF
                - medical management then surgical ligation at 1-3 months
            - if no CCF
                - closure in catheter lab with a coil or device at 1 year
            - there is a slightly increased risk of bacterial endocarditis if left patent

CC7 diseases of the arteries, including aortic dissection

- aortic dissection
    - background
        - diagnosis is challenging
        - mortality rate is 1-2% per hour

- occurs following a tear in the aortic intima with subsequent separation of the tissue within the weakened media by the propagation of blood
    - the aorta is not aneurysmal

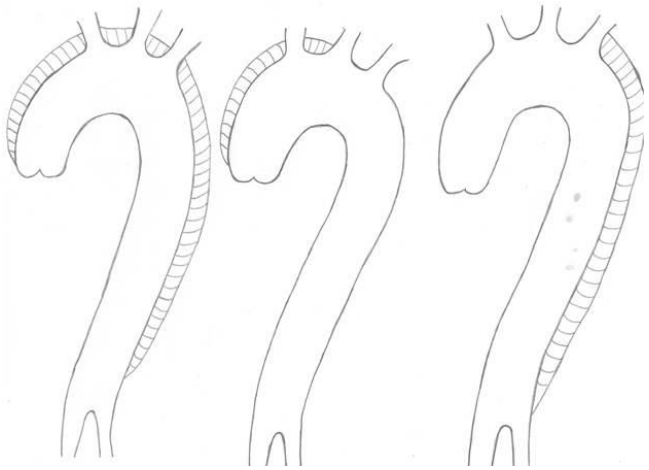
- classification
    - Stanford



https://www.rcemlearning.co.uk/reference/aortic-dissection/#1568112694683-8bf89c2b-c989

- commonest classification
- type A
    - involves the ascending aorta and/or the arch
- type B
    - involves only the descending aorta
    - occurs distally to the origin of the left subclavian artery

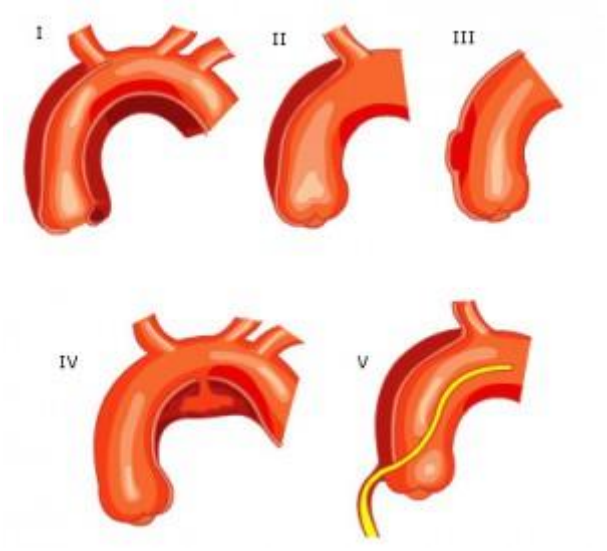
- DeBakeys classification



https://www.rcemlearning.co.uk/reference/aortic-dissection/#1568112694683-8bf89c2b-c989

- 3 types of dissection
- type I
    - involve the entire aorta
- type II
    - only involves the ascending aorta +/- the arch of the aorta
- type III
    - only involves the descending aorta

        - type IIIa
            - involves the aorta just distal to the left subclavian artery, extends proximal or distal to this but largely above the diaphragm
        - type IIIb
            - occurs only distal to the left subclavian artery and may extend below the diaphragm
    - Svensson's



https://www.rcemlearning.co.uk/reference/aortic-dissection/#1568112694683-8bf89c2b-c989

- class 1
    - classic dissection with flap between true and false aneurysm and clot in false lumen
- class 2
    - intramural haematoma
- class 3
    - limited intimal tear with eccentric bulge at tear site
- class 4
    - penetrating atherosclerotic ulcer with surrounding haematoma, usually subadventitial
- class 5
    - iatrogenic or traumatic dissection (e.g. caused by coronary catheter)

- pathophysiology
    - dissection follows a tear in the intimal layer of the aorta with subsequent anterograde or retrograde flow of blood within the outer third of the tunica media
    - thought to occur due to medial degeneration
    - predisposing factors:
        - hypertension
        - Marfan's syndrome
        - Ehlers-Danlos syndrome
        - annuloaortic ectasia and familial aortic dissection

        - bicuspid aortic valve
        - coarctation of the aorta
        - pregnancy
        - Turner's syndrome
        - cocaine abuse
        - giant cell arteritis
        - iatrogenic
- hypertension is the commonest risk factor, present in around 70% of cases
    - causes weakening of the tunica media layer of the aorta
- Marfan's syndrome is associated with a weakened aortic media and progressive aortic dilatation
    - likely to be present in 50% of cases of dissection presenting under the age of 40
- intimal tear
    - the channel created by the dissection process is the false lumen
    - occasionally the blood may re-enter the true lumen through a more distal tear in the intima
    - the majority of intimal tears occur in the ascending aorta due to greater pressure on the aortic wall as it is closer to the left ventricular outflow
- dissection
    - once the dissection process occurs, blood tracks through the media to varying degrees and may dissect down from the aortic root to the bifurcation of the common iliac arteries in seconds
        - typically causes maximal pain at this time
- location of primary aortic tears
    - ascending aorta 70%
    - descending thoracic aorta 15-20%
    - arch of the aorta 10%
    - abdominal aorta <5%
- pathophysiological consequences of dissection
    - rupture into various body cavities
        - ascending aorta
            - haemopericardium
                - syncope and/or sudden death
            - right haemothorax
                - invariably sudden death
        - arch of aorta
            - mediastinal haematoma
            - interatrial septal haematoma
                - cardiac conduction defects
            - compression of pulmonary trunk/artery
        - descending aorta
            - left haemothorax
                - sudden death
            - rarely into oesophagus
                - profuse haematemesis

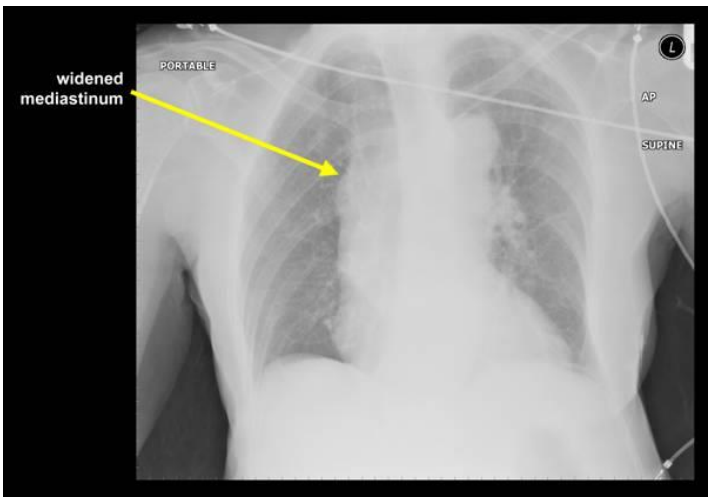
        - abdominal aorta
            - retroperitoneal haemorrhage
                - back pain with shock
            - rarely intraperitoneal haemorrhage
                - shock and acute abdomen
    - occlusion of any of the branch vessels of the aorta with consequent distal organ ischaemia
        - pulse deficits occur
        - smaller branches may be compressed at the point of origin without the dissection progressing within the tunica media
        - the dissection may progress within the media of the branch vessel and the false lumen becomes so large it compresses the true lumen of the vessel
        - occasionally an obstructed vessel may have its blood flow restored because of a re-entry tear into the true lumen or because an intimal flap only intermittently obstructs the origin of the vessel
            - coronary vessels
                - ST elevation myocardial infarction
            - common carotids
                - any type of stroke
            - subclavians
                - acutely ischaemic upper limb
            - coeliac/mesenteric vessels
                - ischaemic bowel
            - renal vessels
                - frank haematuria
            - spinal arteries
                - sudden onset painless paraplegia
    - acute or progressive aortic regurgitation
        - when the dissection extends into or around the aortic valvular support
        - the aortic root dilates so much that the aortic leaflets cannot fully meet during diastole, causing regurgitation through the cusps
        - murmur can be any grade of intensity and may be inaudible if associated haemopericardium
        - patients who survive dissection may present late with cardiac failure
- history
    - pain
        - most common symptom
        - abrupt in onset in 85%
            - almost always maximal pain at onset
        - commonly sharp in nature
        - tearing interscapular pain only in 50%
        - anterior chest in 70-80% of type A dissections

- back pain in 50%
- abdominal pain, throat, neck and extremity pain can also occur
- pain may migrate to limbs or neck, likely due to peripheral extension of dissection
- pain may have resolved by the time the patient attends
    - due to dissection process stopping or pressure relief by re-entry into true lumen

- 5-15% of patients have no pain (particularly elderly patients)
    - their presenting features include:
        - syncope
        - stroke
        - congestive cardiac failure

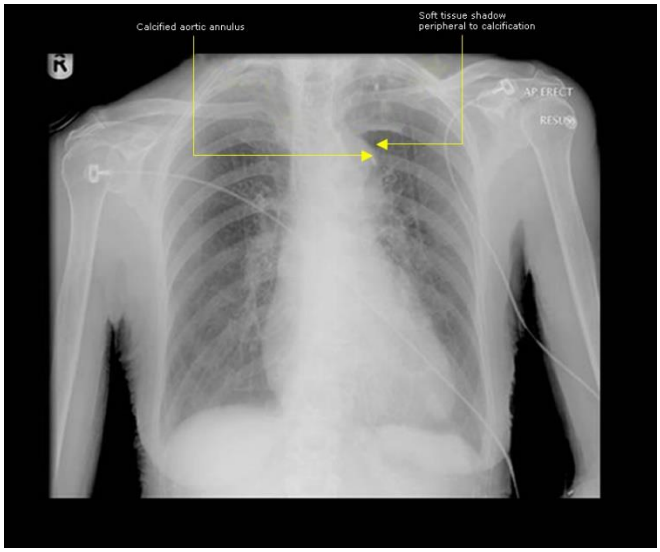
- examination
    - respiratory
        - pleural effusion signs
            - may represent haemothorax
    - cardiovascular
        - signs of haemopericardium
            - pulsus paradoxus
            - faint or absent heart sounds
            - distended neck veins
            - shock
        - signs of aortic root dilatation
            - wide pulse pressure
            - diastolic murmur over aortic area
        - compression of true aortic lumen
            - systolic murmur over any part of the aorta
        - pulse deficits
            - difference of 20mmHg or more in blood pressure between arms
            - weaker central or peripheral pulse compared to contralateral side
            - palpable thrills or audible bruits over pulses
        - if hypotensive
            - determine if secondary to hypovolaemia, pump failure or neurogenic
            - hypovolaemia may be caused by massive haemothorax, loss of blood into extensive aortic wall false lumen, bowel ischaemia with or without haematemesis or retroperitoneal haemorrhage
            - pump failure can be caused by haemopericardium with tamponade, left ventricular dysfunction due to myocardial infarction as a result of a coronary artery dissection, complete heart block following haemorrhage into the interatrial septum
            - neurogenic shock can be caused by spinal cord ischaemia or infarction

        - should be considered when the patient has para- or tetraplegia with bradycardia, hypotension and warm peripheries
    - abdominal
        - to exclude other causes of symptoms such as pancreatitis, perforated hollow viscus, ruptured AAA
- investigation
    - ECG
        - only normal in 30% of patients with dissection
        - findings can include:
            - ST elevation
            - acute ischaemic changes
            - non-specific ST segment and t wave changes
        - if history suggests dissection, diagnosis of acute coronary syndrome should not be made on basis of ECG as treatment may worsen prognosis
    - CXR (usually abnormal)
        - widened mediastinum



https://www.rcemlearning.co.uk/reference/aortic-dissection/#1568112702566-17a3af86-c832

            - present in 60%
        - abnormal aortic contour (50%)
        - soft tissue shadow peripheral to calcified aortic annulus (15%)



https://www.rcemlearning.co.uk/reference/aortic-dissection/#1568112702566-17a3af86-c832

    - represents blood within the false lumen extending around the aortic annulus
  - globular heart
    - haemopericardium
  - pleural effusion (20%)
    - haemothorax
    - usually left sided from descending thoracic aorta
- transthoracic echo
  - must not delay more definitive investigations
  - may identify free intimal flap within the aortic lumen
    - sensitivity 80% for type A, 50% for type B
  - rarely identifies extent of dissection, further imaging required
  - can detect pericardial effusion, aortic regurgitation and degree of aortic valve disruption
- transoesophageal echocardiography (TOE)
  - can be performed in resuscitation room
  - sensitivity of 90-98%
  - can easily delineate the extent of the dissection and visualise the aortic root and valve
  - limited by availability out of hours
  - can cause surges of blood pressure in awake patient, particularly with an inexperienced operator
- CT
  - diagnosis by identifying two distinct lumens with a visible intimal flap
  - can also delineate branch vessel involvement and visualise the entire aorta
  - sensitivity 83-100%
  - also shows pericardial fluid
  - dissection can be missed if there is complete thrombosis of one lumen or similar opacification of both the true and false lumens

- intramural haematoma can be missed if subtle
- MRI
  - requires critically ill patients to be transferred to different monitoring which is magnet proof
  - does not allow quick access to the patient if they deteriorate
  - takes time to acquire images
- aortography
  - invasive and not performed at most hospitals

- management
  - analgesia
    - for comfort and to reduce sympathetic response which can cause progression of dissection
  - blood pressure control
    - active reduction if systolic >120mmHg to reduce progression of dissection
    - requires arterial line
    - IV labetolol (mixed alpha and beta blocker) preferred
      - initial bolus, then infusion titrated to BP
    - vasodilators should not be used alone – cause reflex increase in contractility of left ventricle which potentially will worsen dissection
    - beta blockers should not be used alone – can cause unopposed alpha receptor stimulation with increased peripheral vasoconstriction
    - vasodilators and beta blockers may be helpful if administered together
    - drugs and contraindications
      - beta blockers
        - labetolol
          - 0.25mg/kg (usually 20mg) bolus over 2 minutes
          - further boluses of 20-80mg usually every 10 minutes
          - once BP controlled, continuous infusion at 2mg/minute
          - contraindications
            - cardiogenic shock
            - AV block
            - bradycardia
            - COPD
        - esmolol
          - 200mcg/kg/min loading dose over 1 minute
          - maintenance infusion 50mcg/kg/min for 4 minutes
          - repeat loading dose and increase maintenance by 50mcg/kg/min for 4 minutes if BP still elevated
          - contraindications

        - cardiogenic shock
        - AV block
        - bradycardia
        - COPD
    - metoprolol
        - bolus up to 5mg at 1-2mg/min
        - repeat after 5 minutes to total dose of 10-15mg
        - contraindications
            - cardiogenic shock
            - AV block
            - bradycardia
            - COPD
- vasodilators
    - glyceryl trinitrate
        - titrate 2-10mg/hour
        - contraindications
            - hypotension
            - hypovolaemia
            - HOCM
            - aortic stenosis
    - sodium nitroprusside
        - titrate infusion at rate of 0.5-1.5mcg/kg/min
        - contraindications
            - severe vitamin B12 deficiency
            - hypotension
            - hypovolaemia

- haemopericardium
    - pericardiocentesis must not be performed
        - rapid decompression of the pericardium can restart fresh bleeding with rapidly fatal consequences
            - probably occurs when the pressure gradient between the false lumen and pericardial sac is suddenly increased
- definitive treatment
    - type A
        - urgent transfer to cardiothoracics
        - urgent surgery because of potential to rupture into pericardium with rapidly fatal consequences
        - post-operative inpatient mortality is 26%
    - type B
        - if open surgery not indicated, assessment for presence of complications:
            - persistent intractable pain
            - rapidly expanding aortic diameter
            - development of periaortic or mediastinal haematoma

        - malperfusion of branch vessel organs
        - malperfusion due to aortic lumen compression
      - consideration of endovascular stent-graft
        - successful in 95%
      - for branch vessel occlusion, a stent in the origin of the vessel of percutaneous balloon fenestration can be used
  - pitfalls
    - not adequately treating pain due to concerns that opiates may accentuate hypotension
    - missed diagnosis due to lack of supporting physical signs, normal mediastinum on CXR or ischaemic changes on ECG
    - insufficient time given to history taking, which may provide the diagnosis
    - belief that normal transthoracic echo exclude diagnosis
    - false reassurance due to no difference in bilateral BP
    - allowing BP to remain high whilst awaiting tests/transfer or treating incorrectly with just vasodilator or alpha blocker
- aortic aneurysm
  - background
    - permanent localised or diffuse dilatation of the abdominal aorta to 1.5 times its normal diameter involving all three layers of the vessel wall
    - relatively common
    - rare <50 years
    - 10% rate in men 65-79
    - normal intrarenal diameters in patients under 50 are 1.5cm in women and 1.7cm in men
    - an infrarenal diameter of ≥3cm is considered aneurysmal
  - emergency presentations of AAA include:
    - incidental finding
    - symptomatic or painful aneurysm due to local inflammatory state or acute dissection
    - contained retroperitoneal or compensated rupture (contained leak)
    - uncontained, decompensated rupture with hypovolaemic shock (free rupture)
    - distal embolisation or local thrombosis
    - other atypical presentations
  - causes
    - strongly associated risk factors
      - male gender
      - age
      - smoking
      - hypertension
      - genetic/familial disposition
        - including connective tissue disorders
    - identified and postulated causes
      - atherosclerotic injury and associated degeneration
      - primary connective tissue degeneration
      - inflammatory arteritis with associated aneurysmal degeneration

                - traumatic injury and pseudoaneurysm
                - mycotic injury and pseudoaneurysm
                - aortic dissection-related aneurysmal degeneration
    - pathophysiology
        - true aneurysms involve all 3 layers of the vessel wall and are defined as being >3cm in diameter or an increase >50% from baseline
        - false aneurysms/pseudoaneurysms are deficient in at least one layer
        - fusiform aneurysms have a bulbous shape and are usually true aneurysms
        - saccular aneurysms have a lateral outpouching like a berry aneurysm and are often false aneurysms
        - most AAAs are infra-renal
        - extension above the renal arteries is rare but extension into the iliac arteries is common
        - pathogenesis involves:
            - infiltration of the vessel wall by lymphocytes and macrophages
            - destruction of elastin and collagen in the media and adventitia by proteases, including matrix metalloproteinases
            - loss of smooth muscle cells with thinning of the media
            - neovascularisation
    - acute events
        - pain
            - acute expansion is rarely if ever a cause of pain
            - typically associated with inflammation or dissection
        - rupture
            - contained
                - leak contained within the retroperitoneal space
                - may be relatively haemodynamically stable
            - uncontained
                - free rupture into the intra-peritoneal cavity resulting in shock
        - thrombosis or distal embolization
    - complications
        - death
        - major haemorrhage
        - aortic branch involvement resulting in ischaemia (renal, spinal, pancreatitis)
        - distal embolization
        - rhabdomyolysis
    - clinical assessment
        - palpation of expansile mass
            - two fingers on either side of mass, watching for movement apart with each pulsation
            - may be detectable at 4-5cm depending on body habitus
        - isolated pain in abdomen, epigastrium or back
            - contained leaks typically present with back pain
        - ‘classic’ triad of AAA rupture (not present in all cases, e.g. obesity, hypotension, large retroperitoneal haemorrhage)
            - pain, typically severe and predominantly located in the back

                - signs of circulatory compromise or frank shock
                - presence of pulsatile mass in abdomen
            - atypical presentations in elderly
                - back pain
                    - may mimic renal colic
                - radiation to legs
                    - mimicking sciatica
                - chronic severe back pain
                    - contained rupture
            - tender AAA on palpation is an aortic emergency until proven otherwise
            - massive GI haemorrhage raises suspicion for an aorto-enteric fistula
    - investigations
        - ECG
        - bloods
            - VBG
            - glucose
            - FBC
            - U&E
            - crossmatch
    - imaging
        - US
            - can be performed rapidly at the bedside in an unstable patient
            - can detect aneurysm and free fluid if present (but not confirm rupture)
            - AAA diameter measured as maximum AP diameter in transverse plane
            - imperfect sensitivity (around 95%), 100% specific
        - CT abdomen with contrast
            - best modality if diagnosis is uncertain
            - shows size and extent of AAA, demonstrates leak and complications
            - patients with borderline stability will probably benefit from CT for definitive diagnosis before surgery with ongoing resuscitation in the CT room
        - MRI
            - highly specific and sensitive
            - only suitable for elective cases
        - AXR
            - not recommended but may show mural calcification
            - lateral view may be better as right border of AAA often obscured by vertebral column
    - management
        - rupture
            - 2 large bore peripheral cannulae attached to rapid infuser
            - target systolic of 90mmHg to maintain end organ perfusion
            - crossmatch 6 units and consider activating massive haemorrhage protocol
            - titrated analgesia (e.g. fentanyl, morphine)

                - urgent transfer to operating theatre unless palliation appropriate
                - referral to vascular surgery, anaesthetics and ICU
                - catheter and arterial line (unless it will delay theatre transfer)
            - acute pain
                - urgent referral to vascular surgery
                - acute pain is an emergency even in a stable patient with no evidence of rupture
            - incidental asymptomatic AAA
                - refer to vascular surgeon if surgical candidate (urgency depends on AAA size and presence/absence of symptoms)
                - <2cm normal
                - 2 – 5.5cm diameter and asymptomatic requires regular follow up with US
                - >5.5cm requires operative intervention regardless of symptoms in patients suitable for surgery
            - repair options
                - open repair
                    - only option for ruptured AAA
                    - involves an abdominal or flank incision, vessels above and below the aneurysm are controlled and the aneurysm sac is opened with placement of a synthetic graft
                    - similar mortality to endovascular repair in the long term (8-10 years) for elective repairs
                    - around 20% require subsequent operation
                - endovascular stent grafting
                    - less invasive, lower perioperative morbidity and mortality
                    - involves the intraluminal introduction of a covered stent through the femoral and iliac arteries
                    - can be performed under local anaesthesia
                    - only possible in around 50% of unruptured AAAs
                    - requires long term surveillance due to small risk of aneurysm sac reperfusion and late rupture (endoleak)
                    - 20-30% require secondary intervention during next 6 years
    - prognosis
        - asymptomatic AAA
            - risk of rupture increases with size but any AAA can rupture
            - >6cm diameter has 5 year survival rate of 20% and 50% rate of rupture within 1 year of diagnosis
            - mortality rate decreased by elective repair
        - ruptured AAA
            - 100% fatal unless repaired
            - of those that survive to hospital, mortality for surgical repair is high, up to 50%
            - mortality correlates with age, co-morbidities and hypotension
- aortitis
    - presentation
        - vague symptoms, easily missed

        - pain
        - fever
        - vascular insufficiency
        - elevated levels of acute phase reactants
        - other systemic manifestations
    - causes
        - noninfectious
            - large vessel vasculitides
                - GCA
                    - has aortic involvement in 15%
                    - rare under 50 years
                    - may have widespread vascular inflammation
                - Takayasu arteritis
                    - necrotising large vessel panarteritis
                    - most common in young women, particularly of Asian origin
                    - thought to be secondary to autoimmune process
                    - early stage of fever, malaise, night sweats, weakness, pain
                    - late stage of symptoms related to arterial stenosis, occlusion or dilatation (known as 'pulseless' phase)
                - rheumatoid arthritis
                - SLE
                - ankylosing spondylitis
                - Reiter syndrome
            - medium and small vessel vasculitides
                - Wegener arteritis
                - polyarteritis nodosa
                - Behçet disease
                    - characterised by mucocutaneous ulcers
                    - saccular pseudoaneurysms in the abdominal and thoracic aorta are common
                - relapsing polychondritis
            - idiopathic
                - idiopathic aortitis
                - inflammatory aortic aneurysm
                - idiopathic retroperitoneal fibrosis (periaortitis)
            - radiation induced aortitis
                - most commonly occurs more than 10 years after exposure to a high dose of therapeutic radiation
        - infectious
            - bacterial
                - Salmonella, Staphylococcus
            - syphilis
                - aortic involvement usually evident 5-30 years after initial infection
            - mycobacterial

        - TB
    - viral
        - HIV/AIDS
- imaging
    - multimodality approach usually required
        - CT or MRI usually used

CC8 diseases of myocardium

- myocarditis
    - background
        - inflammation of heart muscle with lymphocytic and fibroblast infiltration and myocyte necrosis
    - causes (HIGAAP)
        - hypersensitivity
        - infectious/infiltrative (haemochromatosis, amyloidosis)
        - giant cell myocarditis
        - autoimmune (SLE, polymyositis, scleroderma, sarcoid)
        - active viral (Coxsackie B, HIV)
        - post viral – lymphocytic (rheumatic fever)
    - history
        - chest pain
        - fatigue
        - shortness of breath
        - palpitations
        - fever
        - malaise
        - arthralgias
    - examination
        - fever
        - tachycardia
        - S3 and S4
        - pericardial rub
        - signs of biventricular failure
        - cardiogenic shock
    - investigations
        - bloods
            - leucocytosis
            - eosinophilia
            - raised ESR
            - raised cardiac biomarkers
            - raised rheumatological markers
        - ECG
            - sinus tachycardia
            - non-specific ST elevation
            - T wave changes
        - echo
            - essential investigation

        - myocardial biopsy
            - for diagnosis based on Dallas criteria
        - serology/PCR
            - enterovirus
            - parvovirus
            - HHV6
            - echovirus
            - coxsackie virus
            - HIV
            - HCV
        - screening for lupus, coeliac disease, TB and other autoimmune conditions as indicated
    - management
        - treatment of underlying cause
        - supportive treatment
            - may include:
                - ACEi
                - beta blockers
                - aldosterone antagonists
                - inotropes/vasopressors
                - IABP or VAD
        - steroids only if secondary to giant cell arteritis
    - complications
        - congestive cardiac failure
        - pulmonary oedema
        - cardiogenic shock
        - dilated cardiomyopathy
        - dysrhythmias
        - recurrent myositis
    - prognosis
        - often excellent
        - depends on clinical presentation, LVEF and pulmonary artery pressure
- cardiomyopathies
    - dilated cardiomyopathy
        - background
            - myocardial disease characterised by ventricular dilatation and global myocardial dysfunction (ejection fraction <40%)
        - presentation
            - usually features of biventricular failure
                - fatigue
                - dyspnoea
                - orthopnoea
                - ankle oedema
        - causes
            - ischaemic
                - following massive anterior STEMI

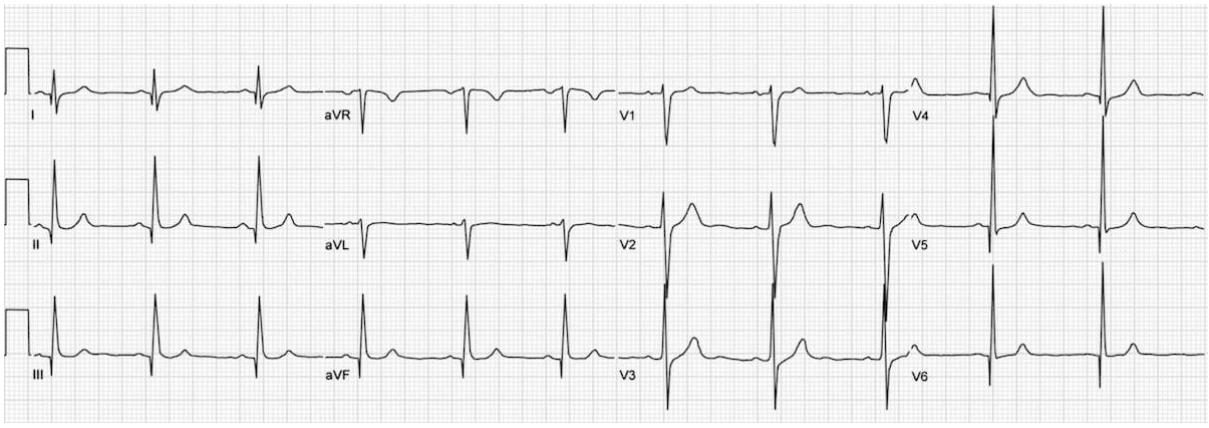
        - due to extensive myocardial necrosis and lack of contractility
    - non-ischaemic
        - idiopathic
            - majority of cases
        - familial
            - up to 25%, usually autosomal dominant, some X-linked
        - infective
            - secondary to viral myocarditis
        - alcoholism
        - toxins
        - autoimmune disease
        - postpartum
- ECG features
    - no specific diagnostic features but usually not normal
    - most commonly changes associated with atrial and ventricular hypertrophy
    - interventricular conduction delays (e.g. LBBB) due to cardiac dilatation
    - reduced voltage QRS complexes
        - particularly in the limb leads
        - due to diffuse myocardial fibrosis
    - abnormal Q waves in leads V1-V4
- investigations
    - CXR
        - cardiomegaly
        - pulmonary oedema
    - echo
        - marked dilatation of the left ventricular cavity
        - reduced systolic and diastolic function
        - may show mitral regurgitation, tricuspid regurgitation and mural thrombus
    - BNP
        - useful in diagnosis, prognosis and management
    - investigations to determine underlying cause
- management
    - aimed at improving cardiac function, treating symptoms and preventing complications
        - loop and thiazide diuretics
            - for symptomatic patients with fluid overload
        - ACEi
            - if left ventricular ejection fraction reduced
        - digoxin
            - if inadequate response to ACEi and diuretics
            - for AF with rapid ventricular rates
        - beta blockers

                - for all patients, shown to improve survival
            - spironolactone
                - shown to improve survival
            - nitrates
                - for diastolic dysfunction and pulmonary congestion
            - anticoagulation
                - for AF, prosthetic heart valve or known mural thrombus
            - implantable cardioverter defibrillator
                - reduces risk of sudden death in high risk patients
            - valve replacement if indicated
        - response to medical therapy may be inadequate and heart transplant or left ventricular assist devices may be required
    - prognosis
        - related to severity of disease at presentation
        - five year survival is around 50%
        - mitral regurgitation or diastolic dysfunction is related to worse prognosis
        - cardiac arrythmia can cause sudden cardiac death
- hypertrophic cardiomyopathy
    - background
        - one of the most common inherited cardiac disorders
        - prevalence 1 in 500
        - main cause of sudden cardiac death in young people
        - caused by mutations in multiple genes coding for sarcomeric proteins
        - inheritance primarily autosomal dominant with variable penetrance
    - structural changes
        - main abnormality is left ventricular hypertrophy occurring in the absence of any causal factors such as hypertension or aortic stenosis
        - degree and distribution of hypertrophy is variable
        - most commonly observed pattern is asymmetrical thickening of the anterior interventricular septum
        - not associated with left ventricular outflow tract obstruction in 75%
        - less common patterns include concentric hypertrophy (20%) and apical hypertrophy (10%)
    - pathophysiology of clinical manifestations
        - dynamic obstruction of the left ventricular outflow tract
        - left ventricular diastolic dysfunction
            - results from impaired relaxation and filling of the stiff and hypertrophied left ventricle
            - often associated with increased filling pressures
        - abnormal intramural coronary arteries with thickened walls and narrowed lumens
        - chaotic, disorganised left ventricular architecture predisposing to abnormal electrical impulses
    - clinical features

- exertional syncope or presyncope
    - suggests dynamic LVOT obstruction +/- ventricular dysrhythmia
    - potential for sudden cardiac death
- symptoms of pulmonary congestion (due to left ventricular dysfunction)
    - exertional dyspnoea
    - fatigue
    - orthopnoea
    - paroxysmal nocturnal dyspnoea
- chest pain
    - may be typical angina pain due to increased demand (thicker myocardial walls) and reduced supply (aberrant coronary arteries)
- palpitations
    - due to SVT or ventricular arrythmias

▪ ECG features

- left ventricular hypertrophy with increased precordial voltages and non-specific ST segment and T-wave abnormalities
- deep narrow 'dagger like' Q waves in lateral (I, aVL, V5-V6) +/- inferior (II, III, aVF)



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- other associated features
    - left atrial enlargement (p mitrale)
        - compensatory left atrial hypertrophy secondary to left ventricular diastolic dysfunction
    - signs of WPW (short PR, delta wave)
        - may be an associated condition
    - dysrhythmias
        - atrial fibrillation and SVTs common
        - PACs, PVCs, VT
    - giant precordial T wave inversions in apical HCM

o restrictive cardiomyopathy

▪ background

- least common form of cardiomyopathy

- occurs in advanced stages of myocardial infiltrative disease (e.g. haemochromatosis, amyloidosis, sarcoidosis)
- diffuse myocardial infiltration leads to low voltage QRS complexes
- atrial fibrillation may occur due to atrial enlargement
- ventricular arrhythmias also common
- infiltration of the cardiac conduction system may lead to conduction disturbance (e.g. bundle branch block, AV block)

▪ ECG features
- low voltage QRS complexes
- non-specific ST segment/T wave changes
- bundle branch blocks
- atrioventricular block
  - 3rd degree AV block may occur in sarcoidosis
- pathological Q waves
- atrial and ventricular dysrhythmias

o septic cardiomyopathy

▪ background
- common feature of severe sepsis
- results in impaired intrinsic cardiac contractility
- 20-60% incidence in first days of ITU admission
- mechanisms are unclear but not due to coronary occlusion

▪ features
- rapid onset
- reversible and leads to full recovery in survivors (usually 7-10 days)
- left ventricular dilatation with normal or low filling pressure
  - due to increased LV compliance and EDV and coexistant EV dysfunction
- global ventricular dysfunction with decreased ejection fraction
  - due to ventricular dilatation despite preserved stroke volume
- absence of regional dysfunction
  - distinct from Takotsubo cardiomyopathy and myocardial ischaemia

▪ investigations
- echo is diagnostic
  - need to exclude other causes of cardiomyopathy
- troponin and BNP are unhelpful and elevated in sepsis

▪ management
- resuscitation
- seek and treat underlying cause of sepsis
  - antibiotics, source control
- fluids if fluid responsive and clinically indicated
  - avoid overload
  - positive fluid balance and raised CVP associated with increased mortality in sepsis
- vasoactive agents
  - inotropes (e.g. dobutamine, adrenaline) for persistent shock

        - noradrenaline
            - improves pre-load and driving pressures for end organ perfusion
    - negative chronotropic agents
        - beta blockers and ivabradine may be useful but evidence lacking
    - circulatory support devices
        - e.g. VA ECMO for severe cases
- prognosis
    - usually resolves in 7-10 days
    - uncertain significance as an independent prognostic indicator
    - impaired intrinsic contractility may be masked by profound vasoplegia, leading to preserved or supra-normal cardiac output
    - dobutamine response predicts improved prognosis in septic shock

- peripartum cardiomyopathy
    - background
        - cardiomyopathy of unknown cause that occurs in the peripartum period
        - rare (1 in 15,000 deliveries)
        - fatal in up to 50%
        - survivors have impaired exercise tolerance and may require transplant
        - postulated causes are viral, autoimmune or an immune reaction to foetal cells
    - definition
        - echo evidence of idiopathic cardiomyopathy:
            - that occurs during a 6 month period peripartum
            - is a new diagnosis
            - other identifiable causes excluded
    - clinical features
        - history
            - failure symptoms
        - examination
            - heart failure
            - pregnancy
    - investigations
        - echo
            - LV dysfunction and normal valves
            - endocardial biopsy
    - management
        - consultation with obstetrician – many therapies are teratogenic or affect foetus
        - reduce preload
            - sodium and fluid restriction
            - diuretics
        - reduce afterload
            - ACEi

        - amlodipine
    - beta blockers
    - anticoagulation if EF <20%
        - if anti-failure treatment fails after 2 weeks consider immunosuppression
    - mechanical support and transplantation may be required
- Takotsubo cardiomyopathy
  - background
    - transient wall motion abnormality of the left ventricular apex associated with severe emotional or physical stress that usually resolves completely
    - produces ischaemic chest pain, ECG changes +/- elevated cardiac enzymes in patients with normal coronary arteries on angiopathy
    - presentation often mimics STEMI
  - Mayo clinic diagnostic criteria
    - transient dyskinesis of the LV apical and/or midsegments
    - regional wall motion abnormalities beyond a single epicardial vascular distribution
    - absence of coronary artery stenosis >50% of culprit lesion
    - new ECG changes (ST elevation or T wave inversion) or moderate troponin rise
    - absence of phaeochromocytoma and myocarditis
  - pathophysiology
    - thought to be related to a combination of sympathetic nervous system activation, microvascular spasm and underlying LVOT obstruction
    - acute stress response leads to catecholamine surge
    - ensuing sympathetic nervous system activation causes microvascular spasm
        - the apical distribution of the left ventricle has the highest density of sympathetic nervous fibres which may explain the characteristic regional wall dyskinesia
    - in at least a third of cases there is LVOT, which increases LV workload, worsening sympathetic nervous system activation and apical dyskinesis
        - patients normally have more severe disease and a worse prognosis
  - clinical significance
    - 90% of cases are in postmenopausal women, usually associated with sudden emotional stress
    - cases in men are more likely to be associated with physical stress
    - difficult to distinguish from STEMI in the ED so STEMI protocol should be followed
    - better prognosis than STEMI with a similar ECG but is not benign
    - treatment is largely supportive and LV function usually spontaneously returns within 21 days

- anticoagulation should be started in patients with large areas of cardiac hypokinesis due to risk of cerebrovascular thromboembolic events

CC9 hypertensive emergencies

- definition
    - accelerated blood pressure – recent increase to very high levels (≥180mmHg systolic and ≥120mmHg diastolic) resulting in target organ damage
        - damage is usually neurological (encephalopathy), cardiovascular or renal
    - where there is no evidence of target organ damage, it is hypertensive urgency rather than emergency and treatment may be more gradual
    - high blood pressure without symptoms is not a hypertensive emergency
    - symptoms such as headache, epistaxis and dizziness are not evidence of acute end organ damage and do not require acute BP reduction
- epidemiology
    - rare, incidence 1-2 cases per million per year
- aetiology
    - may be associated with any cause of secondary hypertension:
        - unilateral renovascular hypertension (renal artery stenosis)
        - renin-secreting neoplasms
        - trauma to the kidneys
        - renal vasculitis
            - e.g. scleroderma, polyarteritis, SLE
        - phaeochromocytoma
        - cocaine abuse
        - drugs
            - monoamine-oxidase inhibitors
            - combined oral contraceptive pill
            - withdrawal of alcohol, alpha stimulants such as clonidine, or beta blockers
        - sodium volume overload and low renin levels
            - e.g. acute glomerulonephritis, primary aldosteronism
        - pre-eclampsia/eclampsia
        - hyperthyroidism/hypothyroidism
- presentation
    - may be asymptomatic
    - may present with symptoms or signs of end organ damage
        - headache
        - fits
        - nausea and vomiting
        - visual disturbance
        - chest pain
        - neurological deficit (e.g. stroke)
        - bleeding due to DIC
        - microangiopathic haemolytic anaemia
- assessment
    - full history including:

        - past medical history
        - systems review
        - drug history including recreational and over the counter
    - full examination including:
        - blood pressure measurements
            - lying
            - standing
            - both arms
        - fundoscopy
            - retinopathy
                - e.g. grade III (flame haemorrhages, dot and blot haemorrhages, hard and soft exudates) to grade IV (papilloedema)
        - cardiovascular examination
            - lying and standing blood pressure
            - signs of cardiac failure or pulmonary oedema
            - carotid or renal bruits
            - left ventricular heave
            - cardiac murmurs
            - third or fourth heart sounds
        - neurological examination
- investigations
    - bloods
        - FBC and clotting
        - U&Es
        - LFTs
        - TFTs
        - glucose
        - consider cardiac enzymes and lipids
    - consider ambulatory blood pressure monitoring
    - urine dip for protein and blood
    - CXR
        - cardiac size
        - signs of cardiac failure
    - ECG
        - left ventricular hypertrophy or left atrial enlargement
    - later investigations may include:
        - CT/MRI of head or kidneys
        - plasma renin activity
        - plasma aldosterone
        - 24 hour urine collection for vanillylmandelic acid and metanephrine levels
        - autoantibodies
- management
    - general measures
        - reduction of blood pressure over 24-48 hours
            - patients usually have altered autoregulation and there may be organ hypoperfusion if pressure drops too fast

        - aim for reduction of around 25% over first 24-48 hours
        - arterial line is useful
        - there may be severe sodium and volume depletion
            - sodium chloride may be needed
    - drugs
        - initially IV
            - labetolol, nicardipine or nitroprusside
        - phentolamine is used for phaeochromocytoma crisis
        - hydralazine can be used for pregnant patients
- prognosis
    - 5 year survival with treatment is >90%
- emergency BP management
    - aim to lower MAP or systolic by no more than 10-20% in first hour
        - except in aortic dissection which requires rapid reduction in systolic to 100-120mmmHg
    - drugs
        - labetolol
            - 20-80mg IV bolus every 10 minutes OR 0.5-2mg/min IV infusion
            - more beta than alpha effects
            - does not change heart rate or cerebral flow
            - rapid onset
            - avoid in COPD, CHF, heart block
            - consider in ACS and ischaemic CVA
        - nitroglycerin
            - start at 5-100mcg/min
            - causes more venous than arterial dilation
            - rapid onset/offset
            - increases coronary flow
            - causes tachycardia
            - use in cardiac ischaemia, LV dysfunction or pulmonary oedema
        - nitroprusside
            - 0.3-0.5mcg/kg/min IV
                - increase by 0.5mcg/kg/min up to 2mcg/kg/min
            - more arterial than venodilation
            - very effective with immediate onset/offset
            - can cause cyanide toxicity and increase heart rate
            - avoid in liver failure, increased ICP and pregnancy
        - phentolamine
            - 5-15mg IV bolus every 5-15 minutes or 0.2-0.5mg/min IV infusion
            - alpha blocker
            - used for catecholamine induced hypertension
        - hydralazine
            - 10-20mg slow IV/IM bolus every 4-6 hours PRN, max 40mg/dose
            - peripheral vasodilator with fall in BP beginning within 30 minutes and lasting 2-4 hours
- NICE guidance for managing hypertension in non-emergency situations
    - definition

        - BP >140/90 and ambulatory BP daytime average or home BP monitoring average of 135/85 or higher
            - assessment of cardiovascular risk should also be done
    - lifestyle interventions
        - diet and exercise
        - reduction in alcohol intake
        - reduction of caffeine
        - reduction of sodium intake
        - cessation of smoking
    - drug treatment
        - ACEi or ARB
            - if type 2 diabetes or under 55 and not black African or African-Caribbean origin
        - calcium channel blocker
            - if 55 or over without type 2 diabetes or black African/African-Caribbean origin and no type 2 diabetes
        - thiazide-like diuretic
            - if CCB not tolerated
        - step 2 treatment
            - if ACEi/ARB insufficient, add CCB or thiazide-like diuretic
            - if CCB insufficient, and ACEi or ARB or thiazide-like diuretic
        - third and fourth medications may be added if needed

CC10 pacemaker function and failure

- definition
    - devices that detect the electrical activity of the heart and stimulate it to contract at a faster rate
- description
    - pulse generator
        - power source
        - battery
        - control circuitry
        - transmitter/receiver
        - reed switch (magnet activated switch)
    - leads
        - single or multiple
        - unipolar or bipolar
- pacemaker code
    - has 5 letters
        - first 3 are antibradycardia functions (always stated)
        - last 2 are related to additional functions
    - 1 – paced chamber
        - 0 = none, V = ventricle, A = atrium, D = dual (A+V)
    - 2 – sensing chamber
        - 0, V, A, D
    - 3 – response to sensing
        - 0 = none, T = triggered, I = inhibited, D = dual (T+I)

    - 4 – rate modulation or programmability
        - 0 = none, P = single programmable, M = multiprogrammable, R = rate modulation in response to minute ventilation or movement
    - 5 – anti-tachycardia functions
        - 0 = none, P = pacing, S = shock, D = dual (P+S) or multi-site pacing
- common pacing modes
    - VVI
        - ventricular pacing and sensing
        - if no electrical impulse sensed then pacemaker will pace at a pre-programmed rate
        - if electrical impulse sensed then pacing inhibited
        - asynchronous pacing
    - VVIR
        - same as above but has rate-adaptive mechanism to match physiological needs of patient
    - DDD
        - both atrium and ventricle sensed and paced
        - if both SA and AV node functioning then pacemaker will just sense
        - if either atrium or ventricle not conveyed, pacemaker will take over
    - DDDR
        - same as above but with rate adaptive mechanism
    - VOO
        - mode that it should be set to for surgery
        - ventricle paced at a pre-programmed rate
        - sensing not interfered with by diathermy or other electromagnetic interference
        - can cause torsades during diathermy due to R on R
- history
    - when pacemaker last checked and result
    - symptoms
        - chest pain, palpitations, blackouts, collapses, orthopnoea, PND, SOBOE, ankle swelling
    - exercise tolerance
    - co-morbidities:
        - IHD
        - hypertension
        - CVA
        - COPD
        - hyperlipidaemia
        - cardiomyopathy
        - valve dysfunction
        - HOCM
        - congenital QT syndromes
    - medications including anti-arrhythmics
    - recent cardioversions
- examination
    - full cardiovascular examination
    - pacemaker site

        - integrity
        - signs of infection
        - relation to any operative site or diathermy
    - signs of heart failure
- investigations
    - electrolytes including magnesium
    - relevant drug levels (e.g digoxin)
    - ECG
        - underlying rhythm and rate
        - pacing spikes
        - electrical activity followed by pacing spike
        - AV synchronicity
    - echo
        - LV and valve function
    - CXR
        - cardiac failure
        - position of pacemaker
        - position of leads
    - angiogram
    - pacemaker interrogation
- malfunctions
    - problems with sensing
        - undersensing
            - when pacemaker fails to sense native cardiac activity
            - results in asynchronous pacing
            - causes include increased stimulation threshold at electrode site (exit block), poor lead contact, new bundle branch block, programming problems
            - ECG findings may be minimal
                - presence of pacing spikes within QRS complexes is suggestive of undersensing
        - oversensing
            - occurs when electrical signals are inappropriately recognised as native cardiac activity and pacing is inhibited
            - inappropriate signals may be large P or T waves, skeletal muscle activity or lead contact problems
            - abnormal signals may not be evident on ECG
            - reduced pacemaker output/output failure may be seen on ECG monitoring if the patient stimulates their rectus or pectoral muscles due to oversensing of muscle activity
    - problems with pacing
        - output failure
            - occurs when a paced stimulus is not generated in a situation when it is expected
            - results in decreased or absent pacemaker function
            - multiple causes, including oversensing, wire fracture, lead displacement, interference

        - failure to capture
            - occurs when paced stimulus does not result in myocardial depolarisation
            - multiple causes, including electrode displacement, wire fracture, electrolyte disturbance, MI, exit block
        - output and capture failure cannot be seen on the ECG if the patient's native heart rate is above the pacemaker threshold, as no activity would be expected
- pacemaker associated dysrhythmias
    - pacemaker-mediated tachycardia
        - re-entry tachycardia in which the pacemaker forms the antegrade pathway with retrograde conduction occurring via the AV node
        - caused by retrograde p waves being sensed as native atrial activity with subsequent ventricular pacing
        - paced ventricular complex results in further retrograde conduction with retrograde p wave generation, forming a continuous cycle
        - results in a paced tachycardia with the maximum rate limited by the pacemaker programming
        - can be terminated by slowing AV conduction (e.g. adenosine or activation of magnet mode)
        - newer pacemakers contain programmed algorithms designed to terminate PMT
        - may result in rate related ischaemia in the presence of IHD
    - sensor induced tachycardia
        - modern pacemakers are programmed to allow increased heart rates in response to physiological stimuli such as exercise or acidaemia
        - sensors may misfire in the presence of distracting stimuli such as vibrations, loud noises, fever, limb movement, hyperventilation or electrocautery
        - misfiring leads to pacing at an inappropriately fast rate
        - the ventricular rate cannot exceed the pacemaker's upper rate limit (usually 160-180bpm)
        - they will usually terminate with the application of a magnet
    - runaway pacemaker
        - potentially life-threatening malfunction of older generation pacemakers
        - related to low battery voltage (overdue pacemaker replacement)
        - pacemaker delivers paroxysms of pacing spikes at 200bpm, which may provoke ventricular fibrillation
        - there may be paradoxical failure to capture, causing bradycardia, because the pacing spikes are very low in amplitude due to the depleted battery, and because at very high rates the ventricle may become refractory to stimulation
        - application of a magnet can be lifesaving but definitive treatment requires replacement of the pacemaker
    - lead displacement dysrhythmia
        - a dislodged pacing lead may float around within the right ventricle, intermittently 'tickling' the myocardium and causing ventricular ectopics or runs of VT alternating with failure of capture

        - if the paced QRS morphology changes from a LBBB pattern (indicating RV placement) to a RBBB pattern (indicating LV placement), this suggests that the electrode has eroded through the interventricular septum
        - CXR helps to confirm diagnosis
    - pacemaker syndrome
        - caused by improper timing of atrial and ventricular contractions resulting in AV dyssynchrony and loss of atrial 'kick'
        - variety of clinical symptoms including fatigue, dizziness, palpitations, presyncope
        - associated decrease in systolic BP >20mmHg during change from native rhythm to paced rhythm
    - Twiddler's syndrome
        - patient manipulation of the pulse generator (accidentally or deliberately)
        - pacemaker rotates on its long axis, resulting in dislodgement of pacing leads
        - can result in diaphragmatic or brachial plexus pacing (e.g arm twitching) depending on extent of lead migration
- management of malfunction
    - contact cardio-technician
    - praecordial thumps (percussive pacing)
    - isoprenaline
    - adrenaline
    - transthoracic external pacing (capture around 80mA)
    - transvenous pacing
    - transoesophageal pacing
- ICD problems
    - shocks delivered whilst patient conscious
        - may also have repeated shocks
        - need to determine whether therapy was appropriate or inappropriate (misidentification of rhythm or noise artefact)
        - should identify and manage pro-arrhythmic electrolyte abnormalities
        - admission usually required unless patient well, investigations normal and only a limited number of shocks sustained
    - syncope
        - bradycardia unlikely
        - VF can result in rapid loss of consciousness and shocks would not be noticed
        - patients with sustained VT may have an element of retrograde amnesia for preceding events
        - admission is usually appropriate
    - dead or dying patients
        - ICD should be electively reprogrammed
        - if not feasible an external magnet should be taped in place across the device

CC11 pericardial disease

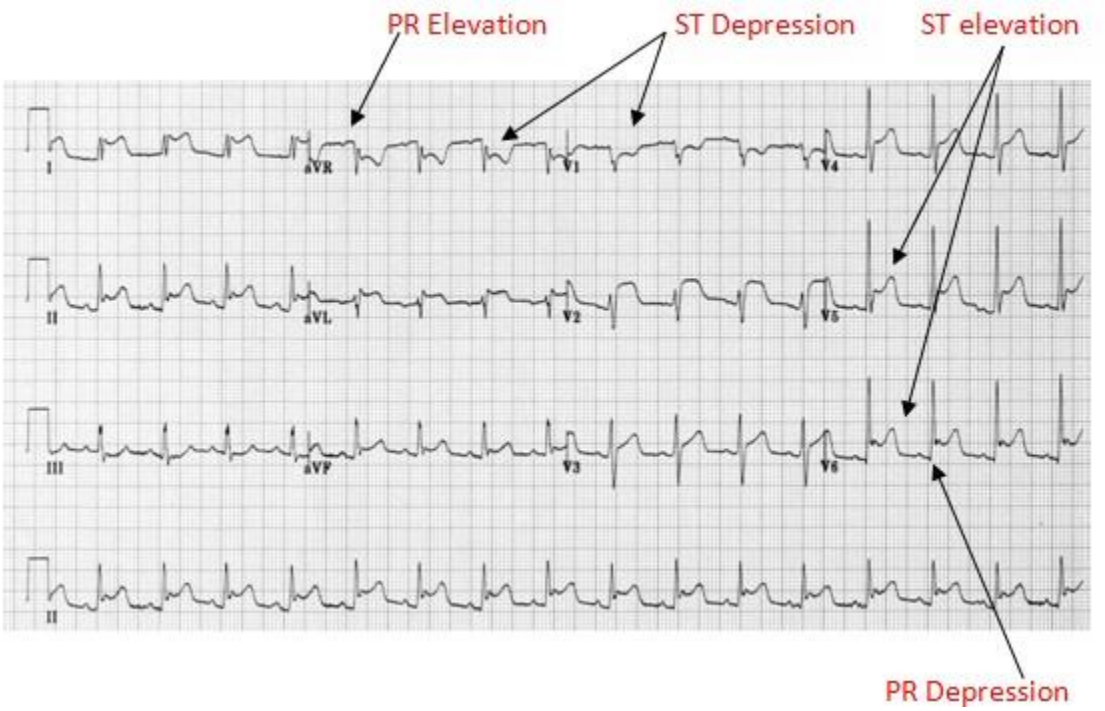
- acute pericarditis
    - definition
        - inflammatory pericardial syndrome with or without effusion
    - causes
        - idiopathic

- 80-90%
- majority are likely to be viral but testing not done as does not alter management

- viral
  - examples include:
    - echovirus
    - enterovirus
    - coxsackie
    - CMV (immunocompromised patients)
    - hepatitis B
    - infectious mononucleosis
    - HIV
- bacterial
  - characterised by purulent effusion
  - commonly direct extension from a pneumonia or empyema
  - patients present more acutely unwell
  - examples:
    - pneumococcus
    - staphylococcus
    - streptococcus
    - mycoplasma
  - tuberculous
    - 1-8% of patients with pulmonary TB
    - more indolent course and non-specific features of fever, malaise, dyspnoea
  - post acute myocardial infarction
    - early (1-3 days):
      - transmural necrosis causing adjacent pericarditis
    - late (1 week to a few months):
      - Dressler's syndrome – autoimmune
  - trauma/post cardiac surgery
    - blunt or penetrating injury
  - neoplastic
    - predominantly from secondary metastatic tumours
  - uraemia
    - pathophysiology unknown
  - inflammatory/autoimmune
    - rheumatoid arthritis
    - SLE
      - 40% get pericarditis at some time
    - scleroderma
  - following chest wall irradiation
    - early:
      - acute illness
    - late:
      - can occur years after exposure
  - drug induced

        - isoniazid
        - hydralazine
- pathophysiology
    - pericardium is composed of two layers:
        - the outer thicker fibrous pericardium
        - the inner visceral/serosal pericardium, made up of a thin layer of mesothelial cells
    - normally the sac between the layers contains 15-50ml of fluid
    - combined thickness of layers should measure less than 2mm
    - pericarditis is caused by inflammation of the pericardial layers associated with varying amounts of pericardial fluid
        - may result in significant pericardial effusion
- history
    - acute onset chest pain
    - classically pleuritic in nature
    - eased by sitting up and leaning forward
    - pain may be anywhere over the anterior chest wall but usually retrosternal
    - may radiate to the arm like ischaemic pain
    - characteristic specific to pericarditis is radiation to the trapezius ridge
        - the phrenic nerve traverses the pericardium and innervates the muscle
- examination
    - pericardial friction rub
        - reported in around a third of cases
        - often dynamic so repeated examination useful if not heard at the outset
        - heard maximally during expiration
        - loudest at left lower sternal edge
        - can be distinguished from pleural rub as it will still be heard when a patient holds their breath
    - fever
        - fever over 38 degrees is high risk and may be associated with bacterial infection
    - clinical features of HIV
    - clinical features of autoimmune disorders
    - clinical features of uraemia
        - non-specific
        - nausea, vomiting, anorexia, itching
    - features of tamponade
- diagnostic criteria
    - at least 2 of:
        - characteristic chest pain
        - pericardial friction rub
        - suggestive ECG changes
        - new or worsening pericardial effusion
- investigations
    - ECG

- characteristic features:



https://www.rcemlearning.co.uk/reference/pericarditis/#1570789448125-2b8d7eb4-1767

    - stage 1 (hours to a few days)
        - diffuse ST elevation (typically concave up)
        - depression of PR segment
    - stage 2 (first week)
        - normalisation of ST and PR segments
    - stage 3
        - diffuse T wave inversions, after ST segments have become isoelectric
    - stage 4
        - normalisation of ECG or indefinite persistence of T wave inversions
- typical changes may occur in around 60% of cases
- dysrhythmias are uncommon and may indicate myocardial involvement
- ECG changes can be confused with benign early repolarisation
    - acute pericarditis is more likely if the ST elevation to T wave height ratio is greater than 0.25

- bloods
    - FBC
        - significantly raised WCC is a poor prognostic sign
    - CRP/ESR
    - U&E
    - troponin
        - raised in 30-70% of patients with acute pericarditis
        - not associated with adverse outcome

        - should be considered to represent myopericarditis and admission and further workup is recommended
    - echo – mandatory
        - large pericardial effusion or tamponade are poor prognostic factors potentially requiring further management
        - up to 60% may have a mild to moderate effusion
    - CXR
        - to look for alternative causes of chest pain
        - may be pneumonia if bacterial pericarditis suspected
        - may be lesions indicative of neoplastic disease
    - CT chest
        - may be performed to look for alternative diagnoses such as aortic dissection or PE
- risk stratification
    - high risk features (should be admitted)
        - fever (>38 degrees)
        - subacute course
        - large pericardial effusion (echo-free space >20mm)
        - cardiac tamponade
        - failure to respond to aspirin/NSAID therapy
        - myopericarditis (elevated troponin)
        - immunosuppression
        - trauma
        - oral anticoagulants
- management
    - activity restriction
        - strenuous activity should be restricted until symptoms have resolved and biomarkers normalised
        - athletes should not compete in competitive sports for at least 3 months following resolution
    - NSAIDs
        - ibuprofen, indomethacin
        - aspirin preferred if pericarditis is a complication of acute MI
        - stop after resolution of symptoms
    - colchicine
        - should be considered in addition to NSAIDs
        - has been shown to reduce symptoms, decrease rate of recurrence and is well tolerated at low doses
        - 0.5-1.2mg/day for 3 months
    - patients not responding to treatment within 1-2 weeks should be admitted for further assessment
    - steroids
        - only if underlying cause is immune mediated or uraemic
        - associated with increased risk of relapse
- complications
    - more common where there is a specific cause
    - acute cardiac tamponade (3.1%)

        - chronic constrictive pericarditis (1.5%)
        - purulent pericarditis associated with tamponade is potentially fatal and requires urgent drainage and IV antibiotics
            - mortality rate 40%
        - relapsing pericarditis occurs in 15-30%
            - some develop chronic relapsing pain
- pericardial effusion
    - causes include
        - idiopathic
        - infectious
            - viral, TB, fungi, parasites, syphilis, bacterial
        - acute MI
        - AKI or CKD
        - malignancy
            - primary and secondary
        - benign tumours
        - hypothyroidism
        - trauma
        - ruptured aortic aneurysm
        - severe chronic anaemia
        - sarcoidosis
        - post-radiotherapy
        - post-cardiac surgery
        - autoimmune diseases
        - drug induced
    - symptoms
        - chest pain, pressure, discomfort
            - may be relieved by leaning forward and is intensified by lying supine
        - lightheadedness, syncope
        - palpitations
        - cough, shortness of breath, hoarseness
        - anxiety and confusion
        - hiccoughs
    - signs
        - classic triad of tamponade
            - hypotension
            - muffled heart sounds
            - jugular venous distension
        - pulsus paradoxus
            - exaggeration of normal respiratory variation in systemic blood pressure
            - can differentiate between effusion and tamponade
        - pericardial friction rub
            - high pitched
            - best heard on expiration with patient upright and leaning forward
            - tachycardia
            - Ewart's sign

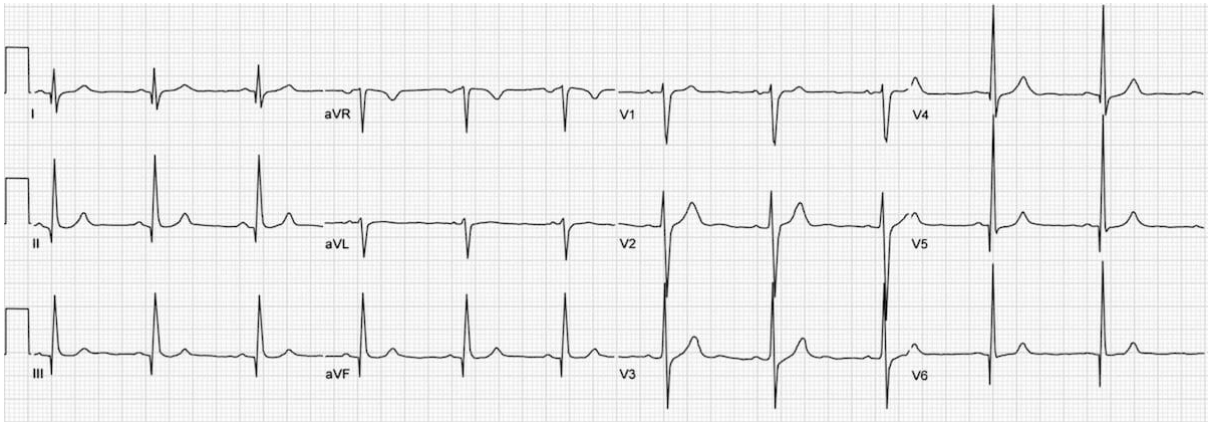
        - dullness to percussion beneath the angle of the left scapula due to compression of the left lung by pericardial fluid
    - investigations
        - bloods
            - FBC
                - raised WCC, cytopaenia as a sign of underlying cancer or HIV
            - U&Es
            - TFTs
            - cardiac enzymes
            - autoantibodies
            - CEA – high specificity for malignant effusion
        - echo
        - CXR
            - globular cardiomegaly with sharp margins in large effusions
        - ECG
            - electrical alternans – alternating high and low voltage complexes
            - raised ST segments with MI or pericarditis
        - MRI
            - can detect as little as 30ml of pericardial fluid
    - management
        - monitoring for small effusions
        - drainage for large effusions if persisting for more than a month or right sided heart collapse
        - admission and drainage for tamponade
        - drainage options include:
            - pericardiocentesis
                - contraindicated in wounds, ruptured ventricular aneurysm or dissecting aortic haematoma
            - surgical approach
                - subxiphoid pericardial window with pericardiostomy
                - pleuropericardial window for persistent effusions, by VATS or thoracotomy
                    - allows drainage of fluid into the pleura where it is more easily absorbed
                - balloon pericardotomy
                - pericardial sclerosis

CC12 sudden cardiac death

- background
    - incidence around 1 in 1000 per year
    - peak incidence in people 45-75
    - in the under 35s, more common in males
    - risk doubles during physical activity
    - predominantly caused by pre-existing congenital cardiac abnormalities
        - can be caused by premature atherosclerotic disease or cocaine use
- causes
    - hypertrophic obstructive cardiomyopathy (HOCM)

        - most common cardiovascular cause
    - dilated cardiomyopathy
    - arrhythmogenic right ventricular cardiomyopathy
    - cardiac ion channelopathies
        - congenital long QT syndrome
        - Brugada syndrome
        - short QT syndrome
    - catecholaminergic polymorphic ventricular tachycardia
    - valvular heart disease (with or without infective endocarditis)
        - aortic stenosis
        - mitral valve prolapse
    - cyanotic heart disease
        - tetralogy of Fallot
        - transposition of the great arteries
    - acyanotic heart disease
        - VSD
        - PDA
    - cardiac arrhythmias
        - WPW
    - coronary heart disease
    - myocarditis
    - myotonic dystrophy
    - Kawasaki disease
    - commotio cordis (traumatic blow to chest wall)
    - cardiopulmonary causes
        - PE
        - aortic dissection
        - ruptured aortic aneurysm
- history
    - preceding symptoms
    - previous medical history
    - circumstances of death/arrest
    - family history
        - including syncope, sudden death, muscle weakness
    - drug history
    - collateral history
- investigations
    - electrolytes and metabolic testing
        - to look for reversible causes of cardiac channel instability
    - markers of cardiac injury
    - autoimmune screen if indicated
    - structural and electrical testing:
        - coronary angiography
            - also rules out congenital coronary anomalies
        - echo
        - resting ECG
        - cardiac MRI
            - unless a clear cause has been identified

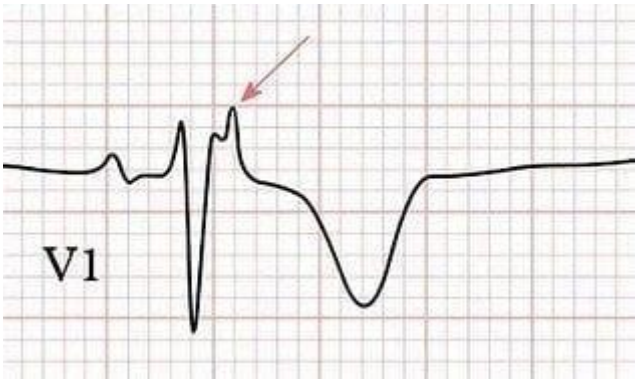
        - treadmill testing
            - uses signal-averaged ECG to look for evidence of late potentials
            - useful in screening for ischaemic cardiomyopathy, subclinical arrhythmogenic right ventricular cardiomyopathy and Brugada syndrome
        - drug provocation testing
            - to unmask a primary electrical cause
                - generally phenotypes of long-QT syndromes, Brugada syndrome and CPVT
    - genetic testing
        - when an inherited phenotype is detected (arrhythmogenic right ventricular cardiomyopathy, Brugada, CPVT, long-QT)
        - to aid diagnosis and family screening
- investigation of first degree relatives
    - focus is on exclusion of known phenotypes/genotypes
    - investigations are confined to an ECG and echo in most cases
- hypertrophic obstructive cardiomyopathy



https://litfl.com/wp-content/uploads/2018/08/ECG-HCM-Dagger-Q-waves-.jpg

    - background
        - unknown aetiology
        - >50% autosomal dominant
        - hypertrophied septum can cause dynamic left ventricular outflow tract obstruction during systole
        - can also get dynamic anterior motion of the mitral valve leaflet towards the septum causing further obstruction
        - causes a pressure overload of LV and diastolic dysfunction
    - history
        - syncope, presyncope, dizziness
        - angina
        - symptoms of heart failure
        - palpitations
        - sudden death
    - examination
        - double or triple apical impulse
        - evidence of heart failure

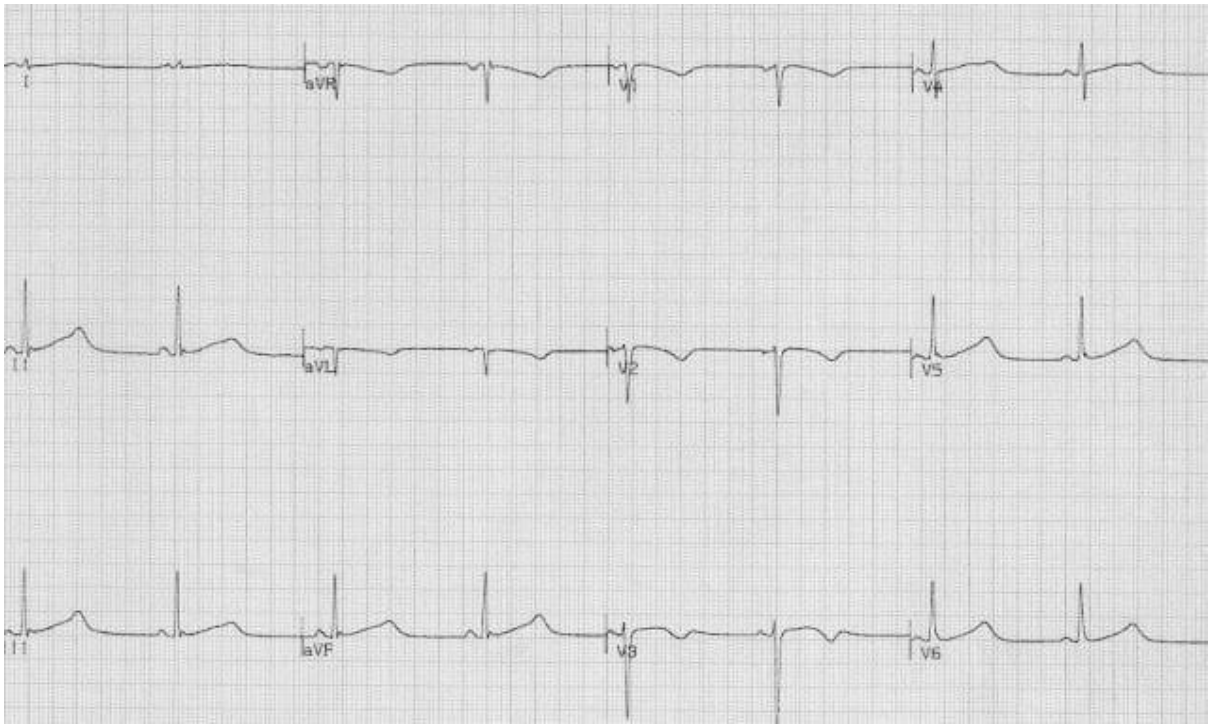
        - jerky pulse
        - S2 may be paradoxically split if very high LVOT gradient
        - prominent a wave on JVP due to decreased RV compliance
        - systolic ejection murmur (due to LVOTO)
            - typically crescendo-decrescendo
            - best heard between the apex and left sternal border, radiates to suprasternal notch but not carotids or neck
        - holosystolic murmur at apex and axilla
            - mitral regurgitation
        - diastolic decrescendo murmur (aortic regurgitation) in 10%
    - investigations
        - ECG
            - LVH criteria
            - deep anterior lateral T wave inversion
            - dagger-like Q waves in infero-lateral leads
        - echo
            - asymmetric septal hypertrophy
            - systolic anterior movement of mitral valve
            - early aortic closure
    - management
        - stop adrenaline
        - volume load
        - beta blockers to slow heart rate and reduce contractility
        - calcium antagonist
        - AV sequential pacing
        - vasoconstrictor without inotropic effect (e.g. phenylephrine, noradrenaline)
        - myomectomy
- arrhythmogenic right ventricular cardiomyopathy
    - background
        - autosomal dominant genetic disorder of the myocardium in which there is fatty infiltration of the right ventricular free wall
        - second most common cause of sudden cardiac death in young people after HOCM
        - prevalence around 1 in 5000
        - diagnosis is difficult and relies on a combination of clinical, electrocardiographic and radiological features
    - ECG features
        - T wave inversion in right precordial leads V1-V3 in absence of RBBB (85% of patients)
        - Epsilon waves (most specific finding, seen in 50%)



https://litfl.com/epsilon-wave-ecg-library/

        - small positive deflection buried in the end of the QRS complex
        - best seen in ST segment of V1 and V2
    - localised QRS widening in V1-V3
    - prolonged S wave upstroke of 55ms in V1-V3
    - ventricular ectopy of LBBB morphology with frequent PVCs (>1000 per 24 hours)
    - paroxysmal episodes of VT with LBBB morphology (RVOT tachycardia)
    - Fontaine bipolar precordial leads can be used to increase sensitivity of epsilon wave detection
        - RA over manubrium
        - LA over xiphoid process
        - LL in standard V4 position (5th intercostal space midclavicular line)
        - leads are named FI, FII, FIII
- clinical features
    - causes symptoms due to ventricular ectopic beats or sustained VT (with LBBB morphology)
    - typically presents with palpitations, syncope or cardiac arrest precipitated by exercise
        - first presenting symptom may be sudden cardiac death
    - over time, surviving patients develop features of right ventricular failure, which may progress to severe biventricular failure and dilated cardiomyopathy
    - usually has family history of sudden cardiac death
- imaging
    - echo
        - may demonstrate dilated, hypokinetic right ventricle with prominent apical trabeculae and dilatation of the RV outflow tract
    - cardiovascular MRI
        - accurately detects structural and functional features
    - histological diagnosis
        - impractical and often at autopsy
- risk assessment
    - high risk of sudden death if:
        - history of syncope due to cardiac arrest
        - recurrent arrythmias not suppressed by anti-arrhythmic drug therapy

        - family history of cardiac arrest in first degree relatives
    - treatment options
        - aim is arrhythmia suppression and prevention of thrombus formation
        - in patients with no high risk features:
            - anti-arrhythmic drugs such as beta-blockers or amiodarone to suppress catecholamine-triggered ventricular arrhythmias
            - most effective drug currently is sotalol
            - warfarin often recommended to prevent thrombus formation due to RV hypokinesis
        - in patients with persistent symptomatic arrhythmias:
            - radiofrequency ablation of conduction pathways may be attempted
            - progression of disease means over half of cases recur
        - patients with any high risk features require urgent insertion of an implantable cardioverter-defibrillator (ICD)
        - heart failure is treated in the usual way with diuretics, ACE inhibitors and anticoagulants
        - in severe cases, cardiac transplantation may be required
- congenital long QT syndrome

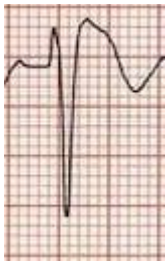


https://litfl.com/qt-interval-ecg-library/

    - background
        - prolonged QT is >450ms (>2 large squares)
        - produces prolonged ventricular repolarisation
        - predisposes to malignant ventricular arrhythmias
        - Romano-Ward syndrome does not have congenital hearing loss, Jervell-Lange-Nielsen syndrome does
        - voltage gated potassium channel gene defect
        - may be sporadic or autosomal dominant

- presentation
  - ranges from no apparent symptoms to tachyarrhythmias resulting in syncope, cardiac arrest, sudden death
    - sinus bradycardia is often seen between episodes
  - may be family history of recurrent syncope or sudden death
- investigations
  - should be considered in all patients presenting with syncope
  - ECG with calculation of QT interval should be performed
  - all other family members must be assessed
- management
  - drug treatment
    - beta blockers
      - usually propranolol
  - if ineffective, selective high left stellate ganglionectomy may be effective
  - permanent pacing in combination with beta blockers may be effective in reducing symptoms
  - for high risk patients, ICD reduces mortality

- Brugada syndrome
  - background
    - ECG abnormality with a high incidence of sudden death in patients with structurally normal hearts
    - diagnosis depends on characteristic ECG and clinical criteria
  - aetiology
    - due to a mutation in the cardiac sodium channel gene
    - can have familial clustering and autosomal dominant inheritance
    - ECG changes can be transient but can be unmasked or augmented by multiple factors:
      - fever
      - ischaemia
      - drugs
        - sodium channel blockers (e.g. flecainide)
        - calcium channel blockers
        - alpha agonists
        - beta blockers
        - nitrates
        - cholinergic stimulation
        - cocaine
        - alcohol
      - hypokalaemia
      - hypothermia
      - post DC cardioversion
  - diagnostic criteria
    - type 1 Brugada changes are the only sort that may be diagnostic



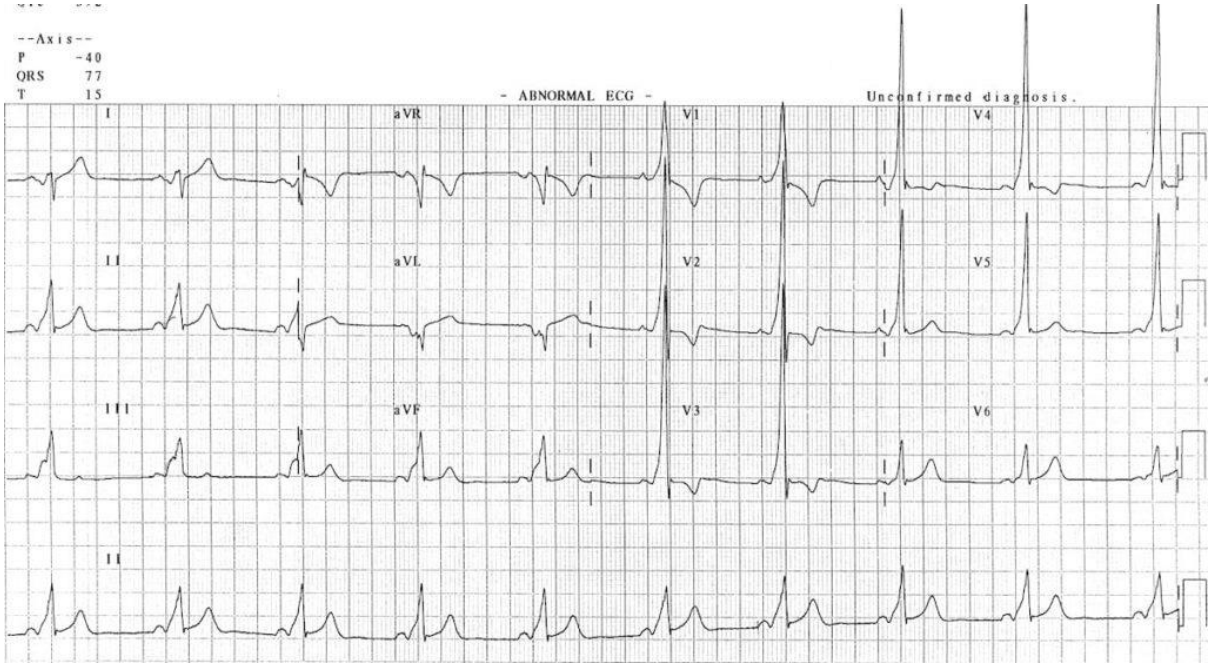
https://litfl.com/what-is-brugada-syndrome/

- cover ST segment elevation of >2mm in >1 of V1-V3 followed by a negative T wave
- must be associated with one of the following clinical criteria to make the diagnosis:
  - documented VF or polymorphic VT
  - family history of sudden cardiac death at <45
  - coved-type ECGs in family members
  - inducibility of VT with programmed electrical stimulation
  - syncope
  - nocturnal agonal respiration

- the other two types are non-diagnostic but may warrant further investigation
  - type 2
    - >2mm saddleback ST elevation
  - type 3
    - morphology of either type 1 or type 2 but with <2mm ST elevation

- management
  - only proven therapy is an ICD
    - quinidine has been proposed when an ICD would be unavailable or inappropriate
  - mortality is around 10% per year if undiagnosed
  - evidence for management is lacking and there may be overinvestigation and overmanagement
  - type 1 patients presenting with suggestive clinical criteria should probably be admitted
  - outpatient management with electrophysiology testing for:
    - asymptomatic patients with type 1 ECG pattern
    - all type 2 and 3 patients

- Wolff-Parkinson-White syndrome



https://litfl.com/pre-excitation-syndromes-ecg-library/

- background
  - pre-excitation syndrome
  - additional or accessory AV pathway (often referred to as the Bundle of Kent)
  - during sinus rhythm the atrial impulse will reach the ventricles via both AV node and accessory pathway
  - accessory pathway conducts impulse faster leading to pre-excitation and short PR interval
  - on reaching the ventricles the pre-excitation impulse is not conducted via the conducting system so ventricular activation is slow (delta wave and T wave abnormalities)
- clinical features
  - palpitations
  - chest pain
  - shortness of breath
  - cardiogenic shock
  - collapse
  - VF arrest
  - AF or SVT
- investigations
  - resting ECG
    - short PR (0.12 sec)
    - T wave abnormalities
    - dominant R in V1 and V2
    - may have inferior q waves, but not diagnostic of MI
  - symptomatic ECG
    - AV re-entrant tachycardia or AF
    - AVRT

        - the re-entry impulse usually travels down the AV node and back up the accessory pathway (delta wave not present); occasionally the re-entry impulse may pass down the accessory pathway and up the AV node (wide QRS tachycardia and delta wave)
    - AF
        - rapid irregular QRS complexes with variable QRS duration
        - very rapid ventricular response leads to cardiogenic shock and VF
- management
    - acute
        - unstable
            - requires synchronised DC shock
        - stable
            - anti-arrhythmics to cause prolongation of accessory pathway
                - sotalol, flecainide, amiodarone, procainamide
        - contraindication for drugs that shorten the refractory period (e.g. digoxin)
        - avoid drugs that increase ventricular rate (e.g. verapamil, lignocaine)
        - drugs that have no effect on the refractory period of the accessory pathway are of no use (e.g. beta blockers)
    - long term
        - radiofrequency ablation

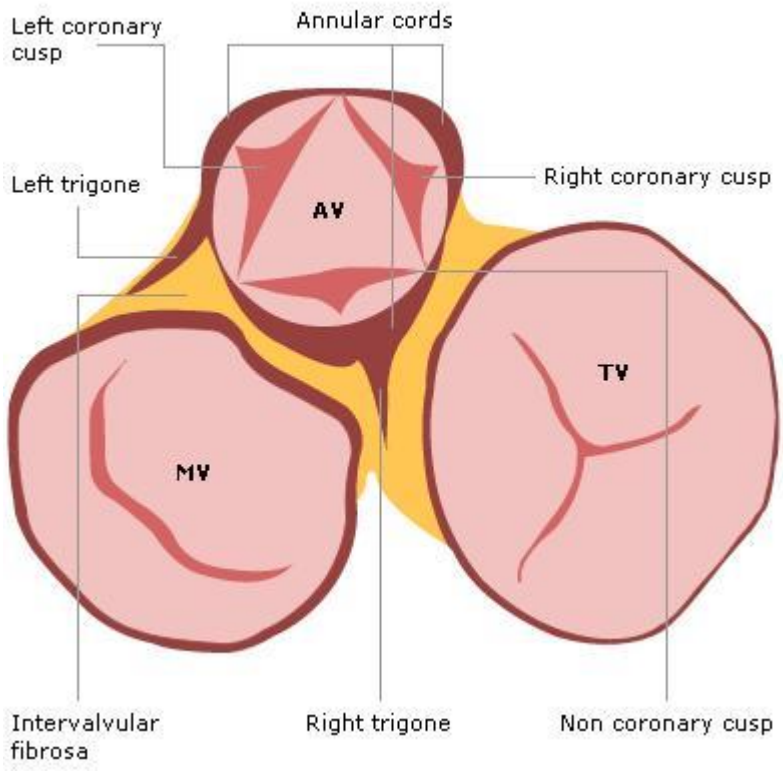
CC13 valvular heart disease

- background
    - three main ED situations
        - hearing a murmur during examination and needing to decide if it is significant
        - patients with known valve problems presenting with signs/symptoms of decompensation requiring onward referral
        - acutely unwell patients with haemodynamic compromise and the need to determine if a heart valve problem is involved
    - major valvular emergencies are:
        - infective endocarditis
        - papillary muscle rupture or flail mitral (posterior) leaflet due to ruptured chordae tendinae
        - prosthetic valve thrombosis/dehiscence
- pathophysiology
    - two cardiac valves on the left side (aortic and mitral), two on the right (pulmonary and tricuspid)
        - mitral and tricuspid valves are known as the atrioventricular (AV) valves
    - closure of the AV valves is helped by the papillary muscles which are attached to the cusps of the valves by the chordae tendinae
        - they act to stop the valves inverting or prolapsing

    - abnormalities of the papillary muscles or chordae tendinae can lead to valve incompetence
        - disruption of normal blood flow places a haemodynamic burden on one or both ventricles
    - valvular stenosis (restricted opening) causes pressure overloading and valvular incompetence (failure of closure) leading to volume overload
    - three main factors for murmurs:
        - forward flow through a narrowed outlet
        - backwards or regurgitant flow through a leaking/incompetent valve
        - high blood flow in high output states such as anaemia, pregnancy, thyrotoxicosis, sepsis, fever
- patient evaluation
    - many murmurs in asymptomatic patients are innocent
        - typically short systolic murmurs heard at the left sternal edge which vary with respiration and are decreased by the patient sitting up
    - description of murmurs
        - where it is maximally heard
        - where it radiates to
        - when in the cardiac cycle it comes (systolic, diastolic, continuous)
        - pitch (high, low, medium)
        - intensity
    - Freeman and Levine loudness grading
        - Grade 1
            - so faint it can only be heard with special effort
        - Grade 2
            - faint but can be heard easily
        - Grade 3
            - moderately loud
        - Grade 4
            - very loud
        - Grade 5
            - extremely loud and can be heard with the stethoscope only just in contact with the skin
        - Grade 6
            - exceptionally loud and can be heard with the stethoscope just removed from contact with the chest
    - types of systolic murmur
        - early systolic
            - acute severe mitral regurgitation
            - tricuspid regurgitation
        - mid systolic
            - physiological due to high output state
            - pathological outflow obstruction
                - aortic stenosis
                - pulmonary stenosis
                - hypertrophic cardiomyopathy
        - pan-systolic

- mitral regurgitation
- tricuspid regurgitation
- ventricular septal defect

- aortic valve disease



https://www.rcemlearning.co.uk/reference/valvular-heart-disease/#1571751404190-ef5294fa-d941

  - aortic stenosis
    - background
      - restricted opening of the valvular cusps causing an obstruction to left ventricular outflow
      - can also be produced by a congenital abnormality above the valve (supra-valvular aortic stenosis) or by sub valvular obstruction due to muscular hypertrophy
      - severe once the valve area has decreased to 1cm^2 or less (normal is 3-4cm^2)
    - aetiology
      - congenital
        - commonest cause in young adults
        - bicuspid or unicuspid valve
      - acquired
        - rheumatic
          - commonest cause worldwide
        - calcific (degenerative)
          - commonest cause in the UK
        - rare causes
          - rheumatoid involvement

        - irradiation
        - obstruction due to infected vegetations
- pathophysiology
    - reduction in aortic valve area leads to a systolic pressure gradient between the left ventricle and the aorta
    - progressive outflow obstruction requires the LV to contract more forcefully to maintain stroke volume
        - eventually results in LV hypertrophy
    - if the mitral valve is functioning normally, the pulmonary circulation is protected and the patient remains asymptomatic
    - in severe disease the ventricle can no longer respond and the LV function becomes abnormal
        - the balance between cardiac output and myocardial muscle oxygen demand can easily be disrupted causing acute decompensation and severe pump failure
- clinical features
    - classic symptoms are:
        - breathlessness
        - chest pain
        - exertional syncope
    - other symptoms include paroxysmal nocturnal dyspnoea, angina and acute MI
    - onset of atrial fibrillation may cause acute progression of symptoms
- clinical and investigation findings
    - pulse
        - slow rising small volume with sustained peak (pulsus parvus et tardus)
        - often absent in the elderly due to loss of aortic compliance
    - cardiac impulse
        - sustained heaving apical impulse with precordial thrill
        - laterally displaced apex beat indicates onset of heart failure
    - auscultation
        - harsh systolic ejection murmur 2nd intercostal space, left sternal edge
            - radiating to the carotids
        - murmur softens and prolongs as severity increases
        - single second heart sound in moderate AS, paradoxical splitting of S2 or soft/obscured by murmur in severe AS
        - fourth heart sound ‘gallop’ rhythm
    - ECG
        - LVH criteria or strain pattern
            - may be absent despite severe obstruction
        - may show RBBB or LBBB
        - atrial fibrillation usually in association with simultaneous mitral valve disease
    - CXR
        - not usually helpful

        - may show normal sized heart and dilated proximal ascending aorta
        - late signs of LV/LA dilatation and pulmonary oedema
        - calcium in the aortic valve of a patient <45 is indicative of AS
    - ED management
        - emergency measures
            - pulmonary oedema
                - diuretics and CPAP to reduce preload and improve ventilatory function
                - avoid nitrates and ACEi as they may cause a drop in afterload and significant BP drop
            - recent excessive diuretics, vasodilator therapy or hypovolaemia may be responsible for acute decompensation and require corrective measures
            - new onset AF
                - digoxin to slow ventricular response and improve stroke volume
            - chest pain
                - beta blockers reduce myocardial oxygen demand and may improve coronary blood flow
            - patients in heart failure may require emergency surgery
            - patients in cardiogenic shock require aggressive medical therapy and emergency surgery
        - other issues
            - patients with AS require antibiotic cover for surgical procedures to protect against infective endocarditis
            - many patients will already have follow up in outpatients if not acutely decompensating
            - prognosis is poor without surgical treatment once symptoms occur
- aortic regurgitation/incompetence
    - background
        - results from failure of the valve to prevent leakage of some of the stroke volume back into the left ventricle from the aorta
        - can occur because of aortic valve leaflet pathology, aortic root disease or a combination of these
        - can be acute or chronic
    - aetiology
        - acute AR
            - aortic dissection
            - infective endocarditis
            - prosthetic valve dysfunction
            - rupture of an aortic valve leaflet (e.g. trauma)
        - chronic AR
            - congenital
                - usually a bicuspid valve or supravalvular stenosis
            - acquired

        - calcific degeneration
        - aortic root dilatation
        - rheumatic fever/previous infective endocarditis
        - rare causes:
            - connective tissue diseases
                - Marfan's
                - Ehlers-Danlos
            - autoimmune diseases
                - rheumatoid arthritis
                - SLE
                - ankylosing spondylitis
            - syphilis
            - appetite suppressant drug Fenfluramine
- pathophysiology
    - regurgitation of blood into the left ventricle during diastole causes volume overload
    - in acute AR there is a sudden increase in the volume of blood in the LV during diastole
        - left ventricular volume can only increase marginally in response to an acute change so left ventricular end diastolic pressure increases sharply
        - LA and pulmonary venous pressures increase and result in acute heart failure
    - in chronic AR there is time for compensation and the LV progressively dilates and hypertrophies to maintain the ejection fraction
        - tachycardia decreases the diastolic filling time and reduces regurgitant volume
        - in the early stages the heart can compensate well with an appropriate increase in cardiac output
- clinical features
    - chronic aortic regurgitation
        - often asymptomatic for years
        - common symptoms
            - awareness of heart beat/palpitations
                - especially at rest
                - due to hyperactive dilated LV
            - chest pain
            - fatigue
        - as the disease progresses
            - heart failure
            - angina (can occur despite normal coronary arteries)
    - acute aortic regurgitation
        - clinical presentation depends on underlying cause
        - if mild, symptoms may relate to underlying cause
- clinical and investigation findings
    - chronic AR

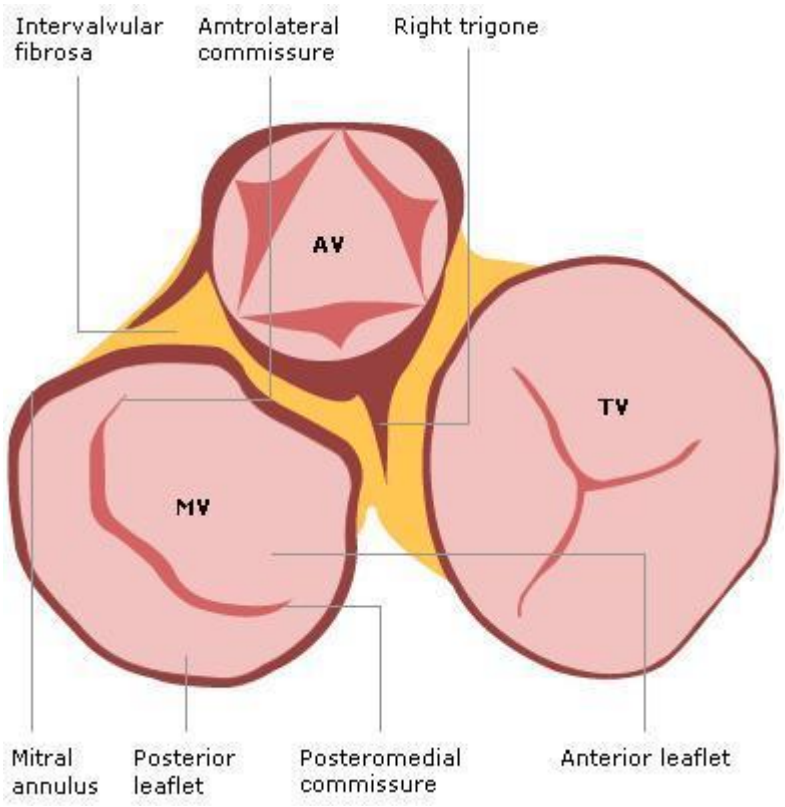
        - pulse
            - rapid rise and quick collapse (water hammer pulse), double impulse, wide pulse pressure
            - Corrigan's sign – visible carotid pulsation
            - Traube's sign – pistol shot sound heard over femoral artery
            - Quincke's pulse – capillary pulsation visible on shining a light through the fingertips
        - cardiac impulse
            - hyperdynamic, may be visible
        - auscultation
            - soft blowing diastolic murmur left sternal edge
                - best heard with patient sitting forward in fully held expiration
            - duration of murmur in diastole correlates with severity of AR
            - Austin Flint murmur – apical diastolic murmur caused by obstruction of mitral flow produced by the partial closure of the mitral valve by the regurgitant jet and rapid rising LV diastolic pressure
                - may be seen in severe AR
        - ECG
            - in moderate/severe disease, LVH with or without strain pattern
        - CXR
            - cardiomegaly with LV prominence and possibly dilated aorta
    - acute AR
        - pulse
            - tachycardia
            - rapid rate of rise of arterial pulse
        - cardiac impulse
            - normal or hyperkinetic
        - auscultation
            - early blowing diastolic murmur
        - ECG
            - non-specific ST changes
            - sinus tachycardia
        - CXR
            - normal heart size and pulmonary oedema
- ED management
    - take blood cultures unless there is an obvious underlying cause
    - immediate specialist review as may require surgery
    - supportive measures to reduce pulmonary venous pressure and increase cardiac output
        - includes vasodilators, intubation and positive pressure ventilation

- inotropic support may be needed but can worsen AR
- nitrates and diuretics have little effect
- IABP is contraindicated

▪ other issues

- patients with AR are at risk of endocarditis and should have appropriate antibiotic prophylaxis

- mitral valve disease



https://www.rcemlearning.co.uk/reference/valvular-heart-disease/#1571751410012-897a33ae-7d58

○ mitral stenosis

▪ background

- mitral valve narrowing restricts blood flow from the left atrium into the ventricle, impairing left ventricular filling
- symptoms may not occur until valve area is reduced to 1-1.5cm^2 (normal 4-6cm^2)

▪ aetiology

- rheumatic heart disease (commonest cause worldwide)
- other rare causes:
  - infective endocarditis
  - calcification of the mitral annulus
  - SLE
  - carcinoid syndrome
  - left atrial myxoma can cause left atrial obstruction and mimic mitral stenosis

▪ pathophysiology

        - obstruction to atrial emptying causes an elevation in left atrial and pulmonary venous pressure
            - leads to reduced lung compliance and breathlessness on exertion
        - reactive pulmonary arterial hypertension causes right ventricular hypertrophy and failure
        - progressive stenosis causes left atrial dilatation and consequent atrial fibrillation which further impairs the function of the atrium
            - left ventricular filling becomes impaired and cardiac output compromised
- clinical features
    - exertional breathlessness, orthopnoea, PND
        - breathlessness on exertion is often the first symptom noticed
    - acute pulmonary oedema
        - hyperdynamic states with an associated tachycardia such as pregnancy, infection, uncontrolled AF and anaemia may worsen symptoms
    - atrial fibrillation
        - onset is associated with a marked deterioration in the patient's state
        - there is risk of left atrial thrombus and systemic embolism
    - haemoptysis
        - used to be the second most common presentation but is rarer now that the disease is recognised sooner
    - fatigue
        - due to reduced cardiac output
- clinical and investigation findings
    - pulse
        - small volume, irregular (usually AF)
    - cardiac impulse
        - 'tapping' apex due to palpable first heart sound (S1)
    - auscultation
        - loud S1 in sinus rhythm
        - opening snap
        - rumbling mid diastolic murmur
        - early diastolic murmur of pulmonary regurgitation (Graham Steell murmur)
    - ECG
        - broad or biphasic p wave best seen in lead 2 indicating LA hypertrophy
        - right axis deviation
        - AF common
        - RV hypertrophy in later stages
    - CXR
        - straightening of the left heart border indicating a dilated LA (double atrial shadow)

        - pulmonary congestion
    - other features
        - mitral facies – pink/purple patches on the cheeks reflecting reduced cardiac output and vasoconstriction
- ED management
    - any cause of tachycardia will result in decompensation
        - symptoms will improve by slowing heart rate and allowing better ventricular filling during diastole
    - close attention to fluid balance
    - antipyretics as appropriate
    - find and treat any underlying infection
    - diuretics may be needed to relieve pulmonary congestion but addressing the shortened diastolic filling caused by tachycardia will be of most benefit
    - rate control
        - beta blockers
        - digoxin
        - calcium channel blockers
    - consideration for cardioversion must take into account significant risk of atrial thrombus and embolization
    - acute haemoptysis is rare but can be significant
        - caused by vessel rupture due to venous congestion
        - may require referral to cardiothoracic surgeon
    - all patients with mitral stenosis and atrial fibrillation should be on long term anticoagulation
        - patients may also present with complications of over anticoagulation

- mitral regurgitation
    - background
        - back flow of blood from the left ventricle into the left atrium during systole
        - causes volume loading of the left atrium and an increased workload for the ventricle to maintain the ejection fraction
        - may be acute or chronic
            - acute MR is a cardiovascular emergency
        - occurs in around 2% of the population
    - aetiology
        - chronic MR
            - rheumatic heart disease
            - LV dilatation secondary to ischaemic heart disease or cardiomyopathy
            - myxomatous degeneration
            - mitral valve prolapse
        - acute MR
            - ruptured chordae tendinae or partial or complete papillary muscle rupture (e.g. due to acute MI, trauma or infective endocarditis)

- pathophysiology
    - during systole, a portion of the ejection fraction regurgitates into the left atrium (the regurgitant volume)
        - can also be expressed as the regurgitant fraction (regurgitant volume/ejection volume)
        - moderate MR is a regurgitant fraction of 30-50%, severe is >50%
    - in acute MR there is sudden volume and pressure overloading of the LA and pulmonary veins leading to acute pulmonary congestion
    - the LV stroke volume increases to maintain cardiac output but in acute MI the ventricle may fail, leading to cardiogenic shock
- clinical features
    - chronic mitral regurgitation
        - with progressive leaking the left side of the heart has time to adapt
        - the LA and LV will both enlarge to cope with the increase in blood volume
        - the LV will hypertrophy to deliver increased stroke volume to maintain cardiac output
        - dilatation of the LA may result in AF and marked symptoms
    - acute mitral regurgitation
        - acutely unwell patient with signs and symptoms of acute pulmonary oedema as well as of the underlying cause
        - urgent echo required to diagnose MR, rule out VSD and assess LV function
- clinical and investigation findings
    - chronic MR
        - pulse
            - tachycardia and AF are common
            - prominent a wave in JVP in SR
        - cardiac impulse
            - diffuse and displaced laterally
            - systolic thrill at apex
        - auscultation
            - pansystolic murmur radiating to axilla and back
            - 3rd heart sound
        - ECG
            - LV and LA hypertrophy
            - AF common
        - CXR
            - increased LA and LV size
            - pulmonary venous congestion
    - acute MR
        - pulse
            - tachycardia
        - cardiac impulse
            - hyperdynamic

        - auscultation
            - pansystolic murmur radiating to the axilla and back
            - 3rd heart sound
            - may be difficult to hear in the acutely breathless and tachycardic patient
        - ECG
            - no changes or acute MI
        - CXR
            - pulmonary oedema with a normal sized heart or minimally enlarged LA
    - management in the ED
        - blood cultures if there is acute MR with no obvious infarct
        - specialist review urgently as may require surgery
        - treat acute MI if identified as underlying cause
        - treat pulmonary oedema
            - may be difficult if patient is in cardiogenic shock
            - intubation and positive pressure ventilation should be considered early
            - CPAP may be helpful
            - reduce preload and afterload with nitrate infusion and ACEi if tolerated
            - diuretics and inotropes may be needed
            - patients with cardiogenic shock from acute MR may benefit from IABP
        - new onset of AF
            - therapy is directed at reducing afterload to reduce LV work and controlling AF
- mitral valve prolapse
    - background
        - prolapse of a portion of the valve leaflets into the left atrium during systole associated with a small amount of regurgitation of blood
        - found in 2-5% of the population
        - more common in women
        - most cases are idiopathic
        - can be acquired secondary to IHD, rheumatic heart disease and hypertrophic cardiomyopathy
    - pathophysiology
        - one or both of the mitral valve leaflets shows fibromyxomatous changes
        - at the end of diastole the valve closes normally, but as the pressure in the LV rises, the leaflet prolapses back into the LA
        - strain on the papillary muscles can lead to mitral regurgitation
    - clinical features
        - often asymptomatic
        - wide variety of associated symptoms such as chest pain, breathlessness and palpitations
        - may progress to clinically significant MR

- increased risk of infective endocarditis and cerebrovascular events
- men, patients over 45 and patients with significant MR at are higher risk for complications

- clinical and investigation findings
  - pulse
    - normal
  - cardiac impulse
    - normal
  - auscultation
    - midsystolic click – a high pitched sound caused by the sudden tensing of the mitral valve apparatus as the leaflet prolapses
  - ECG
    - usually normal
  - CXR
    - no abnormality unless significant MR
- ED management
  - if MVP is diagnosed or suspected, the role of emergency care is to exclude another acute cause for the presenting symptoms
  - otherwise the patient should be referred back to their own GP for further investigation and follow up

- right-sided valve lesions
  - tricuspid regurgitation
    - aetiology
      - congenital
        - Ebstein's anomaly
      - acquired
        - RV dilatation
          - mitral valve disease
          - RV infarction
          - pulmonary hypertension
        - infective endocarditis
          - IV drug abuse
        - Marfan's syndrome
    - clinical features
      - usually asymptomatic unless right heart failure develops
        - oedema, ascites, abdominal pain from liver congestion
      - IV drug users may present acutely unwell with staphylococcal endocarditis
    - clinical and investigation findings
      - pulse
        - AF common
        - large v waves in JVP
      - auscultation
        - soft pansystolic murmur at left sternal edge, louder on inspiration
        - third heart sound (S3) often heard

                - ECG
                    - no specific changes
                - CXR
                    - cardiomegaly
                    - pleural effusion
                - other features
                    - tender enlarged pulsatile liver
            - acute problems
                - tricuspid endocarditis in an IV drug user requires blood cultures and aggressive antibiotic therapy
                    - early surgery may be needed
- prosthetic valves
    - background
        - two main types:
            - mechanical, non-tissue valves
            - porcine, bovine or human tissue valves
        - prosthetic valves can last for decades but patients should attend follow up to detect any deterioration which can progress rapidly
    - valve thrombus and embolization
        - mechanical valves requires lifelong anticoagulation
            - valve thrombosis is often associated with insufficient anticoagulation
        - more common with mitral than aortic valves
        - patient may present with cardiogenic shock, systemic embolization (e.g. cerebral infarct) or sudden death
            - diagnosis should be suspected if patient is known to have a mechanical heart valve or distinctive click is heard on auscultation
            - echo is needed to confirm diagnosis
        - CT should be performed to exclude bleed if there is a cerebrovascular event
        - management is with heparin anticoagulation, thrombolysis, thrombectomy or valve replacement
    - endocarditis
        - most common in the first 2 months post valve surgery
        - usually caused by wound infection or IV line
        - late infections are caused by the same organisms which infect native valves
        - patients with prosthetic valves should have antibiotic prophylaxis
    - prosthetic valves and acute haemorrhage
        - risk of causing thrombosis is outweighed by the risk of ongoing bleeding
        - warfarin should be reversed in consultation with haematology
        - when stable, the patient should receive heparin until warfarin is restarted
- infective endocarditis
    - background
        - increased risk for patients with existing heart valve abnormalities or prosthetic valves
    - presentation
        - patients may present with clear cardiac manifestations or be systemically unwell

        - possible presenting features include:
            - symptoms and signs of infection
            - cardiac involvement
            - immune complex deposition
            - signs of septic emboli
                - may cause focal neurological deficits
        - classical signs (rare)
            - splinter haemorrhages
            - Osler's nodes
            - Janeway lesions
            - Roth's spots
        - conjunctival or buccal petechial haemorrhages are common
    - diagnosis
        - modified Duke criteria
            - two major or
            - one major and three minor or
            - five minor
        - major criteria
            - positive blood culture for infective endocarditis
                - typical consistent microorganism from 2 separate blood cultures (or 3 or 4 separate cultures) – e.g. Strep viridans, Strep bovis, HACEK group
            - evidence of endocardial involvement
                - positive echo (e.g. oscillating intracardiac mass on valve or supporting structures, abscess, partial dehiscence of prosthetic valve)
            - new valvular regurgitation
                - not worsening or change in pre-existing murmur
        - minor criteria
            - predisposition
                - predisposing heart condition or IV drug use
            - fever >38 degrees
            - vascular phenomena
                - major arterial emboli
                - septic pulmonary infarcts
                - mycotic aneurysm
                - intracranial haemorrhage
                - conjunctival haemorrhages
                - Janeway lesions
            - immunologic phenomena
                - glomerulonephritis
                - Osler nodes
                - Roth spots
                - rheumatoid factor
            - microbiological evidence
                - positive blood culture but does not meet major criteria or serological evidence of active infection consistent with IE

- echo findings
    - consistent with IE but do not meet major findings
- management
    - 3-4 sets of blood cultures should be taken from different sites at least one hour apart
    - antibiotics should be discussed with microbiologist prior to starting
    - patients with haemodynamic compromise should be discussed with cardiac surgeons immediately as immediate surgery may be indicated – although mortality is high

DERMATOLOGY

DP1 dermatological manifestations of systemic illness

- pruritus
    - haematological disorders
        - iron deficiency anaemia
        - myeloproliferative disorders (including polycythaemia, leukaemia)
        - monoclonal gammopathy and multiple myeloma
        - lymphoma
    - renal disorders
        - uraemia due to any cause
    - liver disorder
        - cholestasis due to any cause
    - endocrine disorders
        - hyper-or hypothyroidism
- erythema nodosum
    - acute, reactive inflammation of the subcutis, or panniculitis
    - commonly affects young women, presents as symmetric tender, hot, erythematous nodules over the extensor legs
    - usually has a self-limiting course
    - causes and investigations include:
        - infections
            - types
                - bacterial
                    - Streptococcus
                    - Yersinia
                    - Salmonella
                    - Campylobacter
                - viral
                    - Epstein Barr
                - mycobacterial
            - investigations may include:
                - throat swab, antistreptolysin O titre, anti-double-stranded DNA antibodies
                - stool cultures
                - CXR and TB testing

        - sarcoidosis
            - CXR
            - serum ACE inhibitor
            - calcium
        - inflammatory bowel disease
            - history and examination
        - malignancy
            - e.g. leukaemia, lymphoma, post-radiotherapy
            - FBC and film
        - pregnancy
            - history, beta-hCG
        - Behçet syndrome
            - history of oral and genital aphthous ulcers
        - drugs
            - recent commencement (e.g. oral contraceptive pill, tetracyclines, sulphur based drugs, bromides, iodides)
- cutaneous vasculitis
    - infections
        - types
            - bacterial
                - Streptococcal, meningococcal, urinary tract
            - viral
                - hepatitis B/C, HIV
            - mycobacterial
                - TB
    - connective tissue disorders
        - SLE and related conditions
        - rheumatoid arthritis
        - systemic sclerosis, Sjögren's
        - dermatomyositis
        - medium vessel vasculitides (Wegner's, polyarteritis nodosum, Churg-Strauss syndrome)
        - malignancy – haematological
        - drugs – including antihypertensives, antibiotics
        - idiopathic – Henoch-Schönlein purpura
- cutaneous manifestations of specific disease
    - hepatitis C
        - cutaneous vasculitis
        - polyarteritis nodosa
        - porphyria cutanea tarda
        - lichen planus
        - necrolytic acral erythema
    - connective tissue disease
        - lupus
            - malar erythema
            - annular/psoriasiform rash affecting arms and V chest
            - plaques with adherent scale causing scarring

        - dermatomyositis
            - heliotrope rash
            - Gottron papules over interphalangeal/MCP joints
            - macular violaceous or poikilodermatous rash over shoulders/hips
            - calcinosis
        - systemic sclerosis/CREST syndrome
            - calcinosis
            - Raynaud's phenomenon
            - sclerodactyly
            - telangiectasia
            - digital infarcts
        - rheumatoid arthritis
            - rheumatoid nodules
            - linear subcutaneous bands
            - rheumatoid neutrophilic dermatitis
    - malignancies
        - intra-abdominal/lung/lymphoreticular
            - acanthosis nigricans – velvety hyperpigmented thickening extending beyond the flexures and neck to involve lips, palms

DP2 rashes

- red flags
    - fever
    - toxic appearance
    - hypotension
    - mucosal lesions
    - severe pain
    - very old or young
    - immunosuppressed
    - new medication
- description
    - macule
        - not raised
        - not fluid filled
        - flat, circumscribed, coloured
        - <0.5cm
    - papule
        - raised
        - not fluid filled
        - solid
        - <0.5cm
    - vesicle
        - raised
        - clear fluid
        - <0.5cm

    - pustule
        - raised
        - pus filled
        - leucocytes or keratin
        - <0.5cm
    - patch
        - not raised
        - no fluid
        - large macule (flat, coloured)
        - >0.5cm
    - plaque
        - raised
        - no fluid
        - superficially raised, circumscribed solid area
        - >0.5cm
    - nodule
        - raised
        - no fluid
        - distinct large papule
        - >0.5cm
    - bulla
        - raised
        - clear fluid
        - large vesicle/blister or exposed epidermal layer
        - >0.5cm
    - wheal
        - raised
        - oedema
        - firm
        - >0.5cm
- assessment
    - distribution and progression of the lesions
    - recent exposures
        - sick contacts
        - foreign travel
        - sexual history
        - vaccination status
    - examination
        - observations
            - fever or hypotension are concerning
        - fully undress to examine trunk and extremities
        - look at palms, soles, mucous membranes
        - touch the skin with gloved hand to determine if lesions flat or raised
        - press on lesions to see if they blanch
        - rub erythematous skin to see if it sloughs
- differential
    - rash
        - acute generalised exanthematous pustulosis

        - allergic reaction
        - aphthous stomatitis
        - atopic dermatitis
        - chickenpox
        - coxsackie
        - dermatitis herpetiformis
        - erysipelas
        - exfoliative erythroderma
        - impetigo
        - measles
        - miliaria (heat rash)
        - necrotising fasciitis
        - pellagra
        - psoriasis
        - pityriasis rosea
        - scabies
        - seborrheic dermatitis
        - serum sickness
        - smallpox
        - shingles
        - tinea capitis
        - tinea corporis
        - vitiligo
    - vesiculobullous rashes
        - febrile
            - diffuse distribution
                - varicella
                - smallpox
                - disseminated gonococcal disease
                - DIC
            - localised distribution
                - necrotising fasciitis
                - hand, foot and mouth disease
        - afebrile
            - diffuse distribution
                - bullous pemphigoid
                - drug-induced bullous disorders
                - pemphigus vulgaris
                - phytophotodermatitis
                - erythema multiforme major
                - bullous impetigo
            - localised distribution
                - contact dermatitis
                - herpes zoster
                - burn
                - dermatitis herpetiformis
                - erythema multiforme minor
                - bullous impetigo

        - folliculitis
- necrotising rashes
    - necrotising soft tissue infections
        - necrotising fasciitis
        - necrotising myositis
        - necrotising cellulitis
    - purpura fulminans
    - drug rash
    - heparin/warfarin induced skin necrosis
- petechiae/purpura
    - abnormal platelet count and/or coagulation
        - septicaemia
        - ITP
        - haemolytic uraemic syndrome
        - leukaemia
        - coagulopathies
        - HSP
    - hypersensitivity vasculitides
    - primary vasculitides
        - Wegener's
        - microscopic polyangiitis
        - Churg-Strauss syndrome
    - secondary vasculitides
        - HSP
        - connective tissue disorder
            - SLE
        - scurvy
        - hepatitis B/C
    - trauma
- erythematous rash
    - positive Nikolsky's sign
        - febrile
            - staphylococcal scalded skin syndrome (children)
            - toxic epidermal necrolysis/SJS (adults)
        - afebrile
            - TEN
    - negative Nikolsky's sign
        - febrile
            - toxic shock syndrome
            - Kawasaki disease
            - scarlet fever
            - DRESS syndrome
            - cellulitis
            - acute generalised exanthematous pustulosis
        - afebrile
            - anaphylaxis
            - scombroid

- alcohol intoxication
- exfoliative erythroderma
- cellulitis
- drug rash
- erythema multiforme
- dermatitis

DC1 common childhood exanthems

- background
  - 3 main types of rash in children
    - the potentially worrying
      - e.g. petechiae/purpura associated with meningitis
    - named rashes
      - those that can be easily recognised, e.g. chickenpox, measles, HSP, erythema multiforme, ITP, eczema, psoriasis
    - everything else
      - nondescript viral rashes
  - around 70% of presentations of children with fever and rash are viral
- history
  - prodromal symptoms
    - irritability
    - loss of appetite
    - fever
    - malaise
    - headaches
- exanthems and enanthems
  - exanthems
    - eruptive skin rashes associated with a fever or other constitutional symptoms
    - arise from an infectious disease or may be drug related
    - occur less frequently due to vaccination programmes
  - enanthems
    - eruptive lesions on the mucous membranes occurring as a symptom of disease
- historical background
  - original six classical childhood exanthems were identified
    - fourth disease is thought to be a variant of scarlet fever and is no longer used
  - first disease
    - rubeola, measles
      - paramyxovirus
  - second disease
    - scarlet fever, scarlatina
      - Streptococcus
  - third disease
    - rubella, German measles
      - rubella virus

- fifth disease
  - erythema infectiosum
    - parvovirus B19
- sixth disease
  - exanthema subitum, roseola infantum
    - human herpes virus 6B or 7

- clinical assessment
  - distribution of exanthem
    - central or peripheral
    - dermatomal distribution
    - extensor surfaces
    - mucosal involvement
  - appearance
    - colour
    - blanching or non-blanching
    - palpability
    - presence of petechiae
  - type
    - macule

https://www.rcemlearning.co.uk/reference/common-childhood-exanthems/#1568715339435-ee8ca8e5-bf04

      - circumscribed area of change in normal skin colour
      - no skin elevation or depression
      - any size
    - papule

https://www.rcemlearning.co.uk/reference/common-childhood-exanthems/#1568715339435-ee8ca8e5-bf04

- solid raised lesion up to 0.5cm in greatest diameter

- nodule

https://www.rcemlearning.co.uk/reference/common-childhood-exanthems/#1568715339435-ee8ca8e5-bf04

- similar to a papule but located deeper in the dermis or subcutaneous tissue
- different to papules by palpability and depth rather than size

- plaque

https://www.rcemlearning.co.uk/reference/common-childhood-exanthems/#1568715339435-ee8ca8e5-bf04

- elevation of skin occupying a relatively large area in relation to its height
  - often formed by a confluence of papules

- pustule

https://www.rcemlearning.co.uk/reference/common-childhood-exanthems/#1568715339435-ee8ca8e5-bf04

  - circumscribed elevation of skin containing purulent fluid of varying character
  - fluid may be white, yellow, greenish or haemorrhagic
- vesicle

https://www.rcemlearning.co.uk/reference/common-childhood-exanthems/#1568715339435-ee8ca8e5-bf04

  - circumscribed, elevated, fluid-containing lesion less than 0.5cm at its greatest diameter
  - may be intra-epidermal or sub-epidermal in origin

        - bulla

https://www.rcemlearning.co.uk/reference/common-childhood-exanthems/#1568715339435-ee8ca8e5-bf04

            - similar to a vesicle but greater than 0.5cm in diameter
- causes of different types of exanthem
    - maculopapular eruptions
        - measles
        - rubella
        - erythema infectiosum (slapped cheek)
        - exanthem subitum (roseola)
        - Lyme disease
        - pityriasis
        - drug-related eruptions
        - erythema multiforme
    - diffuse erythema with desquamation
        - scarlet fever
        - toxic shock syndrome
        - staphylococcal scalded skin syndrome
        - Kawasaki disease
    - vesicobullous or pustular eruptions
        - diffuse varicella zoster
        - diffuse disseminated gonococcaemia
        - local hand, foot and mouth (coxsackievirus)
        - local herpes zoster
        - Staphylococcal bacteraemia

- specific conditions
    - measles

https://www.rcemlearning.co.uk/reference/common-childhood-exanthems/#1568715344507-4b4a4827-ad59

- background
    - caused by a paramyxovirus
    - occurs in epidemics in winter and spring
    - spread by droplets (or, less commonly, aerosol spread)
    - primary site of infection is the nasopharynx
    - incubation period 7-21 days
    - infectivity usually starts several days before symptom onset and lasts for 4 days after rash appears
- clinical assessment
    - erythematous maculopapular rash beginning on the head
        - cephalocaudad progression
    - clinical case definition
        - fever >38.3° (or felt hot)
        - generalised maculopapular rash lasting for more than 3 days
        - at least one of: cough, coryza, conjunctivitis
    - other associated signs:
        - pinpoint elevations of the soft palate
            - coalesce to cause a reddened pharynx
        - blue-white Koplik's spots on the buccal mucosa opposite the second molar and lasting 1-2 days
- investigations
    - oral fluid or serum for IgM antibody testing
    - can be detected by throat swabs or urine in acute cases
- management
    - notifiable illness – based on clinical suspicion
    - children should be kept off school until 5 days after the appearance of the rash
    - treatment is symptomatic
    - patients should return if symptoms do not resolve in a week or if unusual symptoms develop

- additional treatment options for at risk patients (e.g. immunocompromised, malnourished):
    - prophylactic antibiotics
        - may reduce incidence of complications in areas with high case fatality rates
    - human normal immunoglobulin
        - can prevent or reduce severity of an attack in infants under 12 months, immunocompromised children or pregnant patients (if used within 72 hours of exposure)
    - measles, mumps and rubella (MMR) vaccination
        - indicated in healthy unimmunised or partially immunised children within 72 hours of exposure
    - vitamin A supplements
        - can reduce mortality and pneumonia-specific mortality in children under 2 years
- potential complications
    - otitis media
    - bronchopneumonia
    - laryngotracheobronchitis
    - diarrhoea
    - acute encephalitis
    - sub-acute sclerosing panencephalitis

- scarlet fever
    - background
        - exotoxin mediated disease arising from Group A beta-haemolytic streptococci (GABHS)
        - mainly spread by aerosol or droplets; can also be found in contaminated foods
        - incubation period is usually 2-5 days
        - infectivity period is around 5 days from when the patient is given antibiotics
    - clinical assessment
        - usually begins with sore throat, headache, fever, tender cervical lymphadenopathy and malaise
            - abdominal pain may also occur
        - followed by a confluent erythematous rash with sandpaper-like quality

https://www.rcemlearning.co.uk/reference/common-childhood-exanthems/#1568715344507-4b4a4827-ad59

- other associated features:
    - strawberry tongue

https://www.rcemlearning.co.uk/reference/common-childhood-exanthems/#1568715344507-4b4a4827-ad59

    - Pastia's lines in the flexural folds

https://www.rcemlearning.co.uk/reference/common-childhood-exanthems/#1568715344507-4b4a4827-ad59

    - circumoral pallor
    - pharyngitis
    - desquamation of hands, feet and groin

- associated clinical presentations
    - impetigo or pyoderma
    - bacteraemia
    - septic arthritis
    - erysipelas
    - pneumonia
    - necrotising fasciitis

        - vaginitis
        - pericarditis
        - toxic shock syndrome
- investigations
    - streptococcal antibody test
        - not indicated during acute illness, may confirm previous infection
    - throat swab culture test
        - good quality specimen required
        - gold standard test but results can take 24-48 hours
    - rapid diagnostic tests
        - not routinely recommended for sore throats in the UK
        - specificities are generally high but sensitivities vary and are dependent on swab quality
- management
    - notifiable disease based on clinical suspicion
    - antibiotic treatment for 10 days
        - penicillin V or erythromycin or cephalosporin
    - children should be kept off school for 5 days after antibiotics started
        - if antibiotics started for Strep throat alone they should be kept off school for 24 hours
- associated complications
    - sinusitis
    - mastoiditis
    - peritonsillar abscess
    - pneumonia
    - meningitis
    - osteomyelitis
    - septic arthritis
    - cerebral abscess
    - septicaemia
    - myocarditis
    - toxic shock-like syndrome
    - glomerulonephritis (low risk in UK)
    - acute rheumatic fever (low risk in UK)

- rubella

https://www.rcemlearning.co.uk/reference/common-childhood-exanthems/#1568715344507-4b4a4827-ad5

  - background
    - self-limiting benign illness
    - airborne transmission or droplets
    - incubation period around 2 weeks followed by prodrome of headaches, fever and lymphadenopathy
    - infectivity period from 7 days before to 7 days after appearance of rash
  - clinical assessment
    - characteristic macular rash starting on the face and passing down over the body to the feet
    - fever
    - tender occipital and posterior auricular lymphadenopathy
    - arthralgia
    - respiratory involvement
    - Forschheimer spots
      - pinpoint red macules and petechiae seen on the soft palate and uvula during the rash phase
  - investigations
    - requires laboratory confirmation as difficult to distinguish from other viral illnesses
      - particularly important during pregnancy due to risk to foetus
    - IgG and IgM assays should be used
  - management
    - notifiable disease based on clinical suspicion
    - children should remain off school for at least 5 days after the rash appears

        - women should avoid pregnancy until 3 months after immunisation
    - associated complications (rare)
        - encephalitis
        - hepatitis
        - pericarditis
        - neuritis
        - conjunctivitis
        - orchitis
        - arthralgia or arthritis
        - haemolytic anaemia
        - thrombocytopaenia
    - risks of congenital defects are highest (90%) if infection occurs within the first 12 weeks of pregnancy
- erythema infectiosum

https://www.rcemlearning.co.uk/reference/common-childhood-exanthems/#1568715344507-4b4a4827-ad59

    - background
        - caused by parvovirus B19
        - predominantly spread in respiratory droplets
            - can pass from mother to foetus and in blood transfusions
        - incubation period usually between 4 and 14 days
            - can be as long as 21 days
        - infectivity period starts at exposure and lasts until symptoms appear
    - clinical assessment
        - fever and non-specific symptoms appear early
            - followed 2-3 weeks later by rash and arthropathy
        - classical rash is a 'slapped cheek' appearance and lasts 4 days
            - rash is confluent, erythematous, oedematous with patches or plaques on the cheeks and sparing of the nasal bridge and periorbital areas
            - followed by a maculopapular rash to trunk and limbs
                - can vary in intensity and duration
                - can take on a lacy appearance as it fades
    - associated clinical presentations
        - arthropathy
        - Henoch-Schoenlein purpura

- autoimmune disorders
- myocarditis
- hepatitis
- papular purpuric glove and socks syndrome
- meningitis and encephalitis
- fibromyalgia and chronic fatigue syndrome
- chronic infection in patients with immunodeficiency

- investigations
  - not required unless history of immunocompromise, haemoglobinopathy or pregnancy
  - IgM antibodies appear at 10 days post-infection and are detectable for 2-3 months
  - IgG antibodies appear around 14 days post-infection and remain for life
- management
  - mild self-limiting illness for most children
  - symptomatic treatment including analgesia to help with joint pains
  - transfusion may be required for aplastic crisis
  - IVIG has been used for immunocompromised patients
  - pregnant women in contact with parvovirus B19 should have serological testing and obstetrician referral
- complications
  - transient aplastic crises in those with and without underlying haemolytic illness
  - viral transmission in pregnancy is more likely during the first and second trimester
    - foetal hydrops is most likely to occur in the second trimester

- exanthem subitum (sixth disease)

https://www.rcemlearning.co.uk/reference/common-childhood-exanthems/#1568715344507-4b4a4827-ad59

- background
  - caused by human herpesvirus 6 or 7

        - common benign illness and common cause of fever and febrile seizures in infants
        - incubation period 5-15 days
    - clinical features
        - associated with mild respiratory illness, 3-5 days of fever and cervical lymphadenopathy
        - 10% develop the characteristic rash as the fever disappears
            - commences behind the ears
                - discrete blanching macules and papules surrounded by halos
                - typically lasts 1-2 days
        - other symptoms and signs:
            - palpebral oedema (30% of cases)
            - uvulopalatal junction ulcers
            - erythematous papules on the soft palate (Nagayama's spots) (65% of cases)
            - diarrhoea (68% of cases)
            - cough (50% of cases)
        - prenatal and perinatal infections are rare due to maternal antibodies
    - investigations
        - serological testing not necessary in the majority of cases
    - management
        - presentation with febrile seizure may require admission for observation
        - if clinical course does not present or improve as expected, investigation and management for septic illness may be required
        - reactivation of the virus can occur in transplant patients
    - complications
        - meningoencephalitis
        - hemiplegia
    - the rash can be misdiagnosed as measles
- chicken pox (varicella zoster)

https://www.rcemlearning.co.uk/reference/common-childhood-exanthems/#1568715344507-4b4a4827-ad59

- background
  - spread via respiratory droplets
  - incubation period usually 10-21 days
  - infective from when symptoms first appear until last lesion has crusted over (usually 5-6 days after onset of illness)
- clinical features
  - coryza
  - itchy fluid-filled vesicles which progress over the trunk over 3-5 days
  - fever tends to resolve by day 4
  - it is possible to be infected with no symptoms
- investigations
  - usually not required
  - prolonged fever >4 days should prompt suspicion of complications such as secondary bacterial sepsis
    - blood tests and CXR may be required
- management
  - oral acyclovir may reduce the effects if used within 24 hours of onset of rash in immunocompetent children
    - it can reduce number of lesions and duration of fever but not complications such as pneumonia, so is not recommended routinely
  - varicella zoster immunoglobulin (VZIG) for neonates whose mothers develop the rash 7 days before or after delivery to reduce risk of severe neonatal varicella
- complications
  - secondary streptococcal or staphylococcal infections
  - pneumonia
  - bacteraemia
  - encephalitis
    - neurological complications can occur without preceding rash
  - congenital varicella syndrome
    - low if maternal infection is before 20 weeks' gestation
    - associated with shortened limbs, skin scarring, cataracts, growth retardation
  - ibuprofen is not recommended for fever control due to risk of necrotising fasciitis
    - however, the absolute risk increase is tiny and in unwell patients in hospital a risk benefit assessment should be made

- Gianotti Crosti syndrome
  - papular acrodermatitis
  - self-limiting illness peaking between 1 and 6 years
  - associated with a range of viruses and bacteria
    - viral
      - Epstein Barr
      - hepatitis A, B, C

        - cytomegalovirus
        - human herpesvirus 6
        - Coxsackie A16, B4, B5
        - rotavirus
        - parvovirus B19
    - bacterial
        - Bartonella henselae
        - beta-haemolytic streptococci
        - Borrelia burgdorferi
        - mycoplasma pneumonia
- clinical features
        - low grade fever
        - diarrhoea
        - tonsillitis
        - lymphadenopathy
    - more common in children with atopic dermatitis
    - association with immunisation
    - rash

https://www.rcemlearning.co.uk/reference/common-childhood-exanthems/#1568715344507-4b4a4827-ad59

        - papular or papulovesicular
        - symmetrically distributed on the face, buttocks and upper and lower extensor surfaces of the extremities
        - trunk is usually spared
        - lesions can last from 5 days to 12 months
            - most heal within 2 months
- investigations
    - rarely indicated unless diagnostic uncertainty
- management
    - rarely required
    - may need antihistamines for pruritus
- complications
    - rare
    - can include chronic liver disease secondary to hepatitis B infection

DC2 cutaneous drug reactions

- types of hypersensitivity reaction
  - Type I
    - IgE mediated
    - results in urticaria, angioedema and anaphylaxis
    - often caused by proteins
  - Type II
    - cytotoxic reaction
    - causes haemolysis and purpura
    - caused by penicillin, cephalosporins, sulphonamides and rifampin
  - Type III
    - immune complex reactions
    - cause vasculitis, serum sickness and urticaria
    - can be caused by salicylates, chlorpromazine, sulphonamides
  - Type IV
    - delayed type reactions with cell-mediated hypersensitivity
    - results in contact dermatitis, exanthematous reactions, photoallergic reactions
    - most common reactions, usually caused by topical applications
    - not dose dependent
    - usually begin 1-3 weeks after medication is started
- diagnosis
  - requires careful history of medications, including over the counter, herbal and 'natural' preparations and illicit drugs
  - consider foods such as shellfish or strawberries for urticaria
  - consider viral infection
- allergic patterns defined by NICE
  - immediate
    - developing within 1 hour
    - anaphylaxis, urticaria, exacerbation of asthma
  - non-immediate without systemic involvement
    - 6-10 days after first drug exposure or within 3 days of second exposure
    - widespread red macules or papules (exanthema-like) or fixed drug eruption
  - non-immediate reactions with systemic involvement
    - 2-6 weeks after first drug exposure or within 3 days of second exposure
    - drug reaction with eosinophilia and systemic symptoms (DRESS) or drug hypersensitivity syndrome (DHS)
    - widespread red macules, papules or erythroderma, fever, lymphadenopathy, liver dysfunction
  - toxic epidermolysis
    - 7-14 days after first drug exposure or within 3 days of second exposure
  - Stevens-Johnson syndrome
    - 7-14 days after first drug exposure or within 3 days of second exposure
  - Acute generalised exanthematous pustulosis (AGEP)
    - 3-5 days after first drug exposure
    - characterised by widespread pustules, fever, neutrophilia
- types of eruptions

- acneform lesions
  - tend to be over upper body rather than face with no comedones
  - typical drugs:
    - corticosteroids
    - halogens
    - haloperidol
    - hormones
      - including anabolic steroids and progestogen contraceptive pills
    - isoniazid
    - lithium
    - phenytoin
    - trazodone
- acute generalised exanthematous pustulosis (AGEP)

https://dermnetnz.org/topics/acute-generalised-exanthematous-pustulosis/

  - acute fever and generalised scarlatiniform erythema
    - small, sterile, non-follicular pustules
    - appears like pustular psoriasis
  - mostly caused by antibiotics, often in first few days
  - other causes include:
    - viral infections
    - mercury exposure
    - UV radiation
  - resolves spontaneously and rapidly
    - fever and pustules last 7-10 days
    - followed by desquamation over a few days
  - drug causes
    - beta-lactam antibiotics
    - macrolides
    - others (e.g. paracetamol, furosemide, phenytoin, ranitidine, simvastatin)
- alopecia
  - drug causes
    - ACEi

- allopurinol
- anticoagulants
- beta-blockers
- hormones
- phenytoin
- methotrexate

- bullous pemphigoid

https://dermnetnz.org/topics/bullous-pemphigoid/

  - may occur with various drugs including penicillins and furosemide
- erythema nodosum

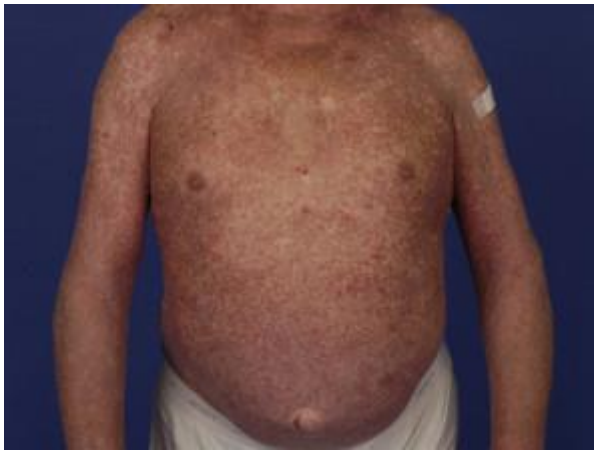
https://dermnetnz.org/topics/erythema-nodosum/

  - most common with oral contraceptives
  - can occur with halogens, penicillin, sulphonamides, tetracyclines
- erythroderma
  - drug causes include allopurinol, anticonvulsants, lithium, nitrofurantoin, omeprazole, phenytoin, St. John's wort, sulphonamides
- fixed drug eruptions

https://dermnetnz.org/topics/fixed-drug-eruption/

- delayed type IV hypersensitivity reaction
- when lesions recur in the same place when the same drug is given
- subsequent flares can be more severe
- circular, violaceous, oedematous plaques
    - resolve with macular hyperpigmentation
- latent period half an hour to eight hours after drug taken
- usual sites are hands, feet and genitalia; perioral and periorbital lesions may occur
- drug causes include:
    - anticonvulsants
    - aspirin
    - NSAIDs
    - benzodiazepines
    - ciprofloxacin
    - doxycycline
    - loratadine
    - metronidazole
    - oral contraceptives
    - penicillins
    - phenytoin
    - sulphonamides
    - tetracyclines

- lichenoid reactions



https://dermnetnz.org/topics/lichenoid-drug-eruption/

        - similar in appearance to lichen planus
        - may have marked pruritus
        - can take weeks to months to disappear, more for nails
        - steroids can help
        - drug causes include:
            - amlodipine
            - beta-blockers
            - enalapril
            - furosemide
            - PPIs
            - sildenafil
            - tetracycline
    - photosensitivity
        - common causes
            - ACEi
            - amiodarone
            - amlodipine
            - diltiazem
            - furosemide
            - quinolones
            - sulphonamides
            - tetracyclines
            - thiazides
- management
    - remove offending drug
    - note adverse reaction
    - antihistamines may give symptomatic relief
    - corticosteroids may be helpful for DRESS
    - referral to specialist drug clinic:
        - suspected anaphylactic reaction
        - severe non-immediate cutaneous reaction
            - e.g. DRESS, Stevens-Johnson Syndrome, toxic epidermal necrolysis

        - suspected beta-lactam antibiotic allergy if patient has a condition that can only be treated by these or is likely to need them frequently in the future
    - prognosis
        - most cases resolves in 10-14 days
        - with exanthematous eruptions mild desquamation occurs as rash resolves
    - red flags
        - systemically unwell
        - extensive rash
            - can lead to serious exfoliative dermatitis
        - detachment of skin
        - involvement of mucous membranes and genitalia

DC3 eczema

- background
    - atopic eczema is common with increasing prevalence
    - affects 10-30% of children and 2-10% of adults
    - can present at any age but more commonly starts in childhood
    - increased prevalence if parents affected
        - 80% where both parents affected
        - 60% where one parent affected
    - increased rate in urban areas, smaller families and higher socio-economic classes
- triggers
    - environmental
        - irritants
            - soaps and detergents
        - skin infections
            - Staphylococcus in particular
        - contact allergens
        - extremes of temperature and humidity
            - most patients improve in summer and are worse in winter
            - sweating can provoke an exacerbation
        - abrasive fabrics
        - dietary factors
            - in about 50% of children but much less in adults
        - inhaled allergens
            - house dust mites
            - pollens
            - pet dander
            - moulds
    - endogenous
        - probably genetic mutations affecting the production of filaggrin
            - a protein critical to conversion of keratinocytes to the protein/lipid squames that compose the stratum corneum
            - there is increased sensitivity to environmental triggers
        - hormonal changes in women
        - stress
- diagnosis

        - must have an itchy skin condition (if no itch it is unlikely to be eczema) plus three or more of:
            - itchiness in skin creases such as elbow folds, behind knees, front of ankles, around neck
        - history of asthma or hay fever (or in first degree relative in children under 4)
        - general dry skin in preceding year
        - visible flexural eczema
        - onset in first 2 years of life
- investigations
    - rarely required
    - scoring scales can be used to assess severity
        - patient-oriented eczema measure (POEM)
        - children's dermatology life quality index (CDLQI)
        - infants' dermatitis quality of life index (IDQOL)
        - dermatitis family impact (DFI) questionnaire
- management
    - education about condition, treatments and quantities of topical treatments
    - information on how to recognise flares
        - increased dryness, itching, redness, swelling, general irritability
    - how to recognise signs and symptoms of bacterial infection
        - weeping, pustules, crusts, failure to respond to therapy, fever, malaise
    - psychosocial support
    - identification and avoidance of provoking factors
    - short nails
    - avoidance of soaps and detergents – replace with emollient substitutes
    - keep skin hydrated
    - emollients should be used even when skin is clear
- emollients and moisturisers
    - best applied when skin is moist but should also be applied at other times
    - as liberally and frequently as possible
        - combination of cream, ointment, bath oil, emollient soap substitute
    - should be every four hours or at least 3-4 times/day
    - need to be prescribed in large quantities (500g/week for adults, 250g/week for children)
    - anyone using paraffin based emollients (also possible risk for paraffin-free emollients) must be advised not to smoke or go near naked flames and be warned about easy ignition of clothing, bedding, dressings
- topical steroids
    - mild for face and flexures, potent for adults with discoid or lichenified eczema or scalp/limb/trunk eczema
    - advised once or twice/day
    - mild potency for face and neck
        - can used moderate potency for severe flares for 3-5 days
    - moderate or potent for short periods only (7-14 days) in vulnerable sites such as axilla/groin
    - topical steroid withdrawal can lead to red skin, burning or stinging, itch, skin peeling and oozing, papules, pustules, erosions, excessive sweating

        - more common with inappropriate long term use of moderate to potent steroid
        - can be mild and short lived or last months or years
- bacterial infection
    - emollient antimicrobial preparations can help prevent infection
    - oral antibiotics for moderate to severe infection (14 day course)
    - NICE recommended options if not systemically unwell
        - topical fusidic acid 2% TDS for 5-7 days
        - flucloxacillin first choice oral antibiotic
            - clarithromycin in penicillin allergy
- lichenification
    - from repeated scratching
    - initially treated with potent corticosteroid
    - bandages with ichthammol paste or other substances may be used
- exudative eczema
    - requires potent corticosteroid and treatment of any infection
    - potassium permanganate solution may be used
- eczema herpeticum

https://dermnetnz.org/topics/eczema-herpeticum/

    - disseminated viral infection most often as complication of eczema
    - fever and clusters of itchy blisters or punched out erosions
    - cause is usually herpes simplex 1 or 2
    - clinical features
        - starts with clusters of itchy and painful blisters
            - most commonly on face or neck
            - new patches form and spread over 7-10 days, may rarely be widely disseminated
        - unwell patient with swollen local lymph nodes
        - blisters
            - monomorphic – appear similar to each other
            - may be filled with clear fluid or thick purulent material
            - often blood stained (red, purple, black)
            - new blisters have central dimples (umbilication)
            - may weep or bleed

                - older blisters crust over and form erosion
                - lesions heal over 2-6 weeks
                    - may have long term scarring if skin damage occurred
            - when severe, may affect multiple organs including eyes, brain, lung and liver
        - diagnosis
            - clinical +/- viral swabs of fresh blisters, viral culture or PCR
        - management
            - dermatological emergency
            - requires same day dermatology referral
            - oral acyclovir 400-800mg five times/day for 10-14 days or until lesions heal
                - or valaciclovir 1g BD if available
                - antivirals may prevent need for admission
                - IV if unable to take tablets or progressing despite oral treatment
            - antibiotics for secondary bacterial infection
            - ophthalmology review if eye or eyelid involvement seen or suspected

DC4 erythroderma

- background
    - intense and widespread reddening of the skin due to inflammatory skin disease
    - rare
    - can occur in all ages and races
    - 3 times more common in males
    - reason for occurrence is unclear, with pathogenesis involving interaction between keratinocytes/lymphocytes and adhesion molecules/cytokines causing dramatic increase in turnover of epidermal cells
- causes and associations
    - 30% idiopathic
    - drug eruption
    - atopic dermatitis
    - psoriasis
    - pityriasis rubra pilaris
    - other forms of dermatitis
        - e.g. contact dermatitis, venous eczema
    - blistering diseases like pemphigus and bullous pemphigoid
    - Sézary syndrome (form of cutaneous T-cell lymphoma)
    - severe rare congenital ichthyotic conditions
    - can be a sign of systemic disease:
        - haematological malignancy
        - internal malignancy (e.g. rectal, lung, colon, prostate)
        - graft-versus-host disease
        - HIV
- clinical features
    - may be preceded by morbilliform (measles-like) eruption, dermatitis or plaque psoriasis
    - generalised erythema developing rapidly in acute cases or over weeks to months in chronic cases
    - erythema and oedema or papulation affect at least 90% of the skin surface

    - signs and symptoms
        - warm skin
        - itch (may be intolerable) and ensuing lichenification from scratching
        - ectropion from eyelid swelling
        - scaling 2-6 days after onset of erythema
            - fine flakes or large sheets
        - thick scaling on scalp with varying degrees of hair loss including complete baldness
        - yellowish, diffuse keratoderma on palms and soles
        - dull, ridged, thickened nails or onycholysis/shedding
        - swollen lymph nodes
    - clues to underlying cause
        - serous ooze with unpleasant smell suggests atopic erythroderma
        - circumscribed scaly plaques at elbows and knees suggests psoriasis
        - islands of sparing, follicular prominence, orange hue to keratoderma suggest pityriasis rubra pilaris
        - subungual hyperkeratosis, crusting on palms and soles and burrows suggest crusted scabies
- complications
    - hypothermia
    - electrolyte abnormalities and dehydration
        - fluid loss through non-intact skin
    - high output heart failure
    - secondary skin infection
        - impetigo, cellulitis
    - pneumonia
    - hypoalbuminaemia from protein loss and increased metabolic rate
        - leads to oedema
- diagnosis
    - may have anaemia, WCC abnormalities and eosinophilia
        - marked eosinophilia should raise suspicion of lymphoma
    - skin biopsies may be required but may be non-specific
    - direct immunofluorescence if autoimmune or connective tissue disease suspected
- management
    - may be life-threatening
    - discontinue all unnecessary medications
    - monitor fluid balance
    - temperature control
    - maintain skin moisture with wet wraps, dressings, emollients, mild topical steroids
    - antibiotics for bacterial infection
    - antihistamines may help with itch
    - specific treatments if cause identified
- prognosis
    - good if underlying cause can be identified and removed
    - may recur or persist

DC5 infections of the skin and soft tissues

- impetigo
    - background
        - caused by Staph aureus or Group A beta haemolytic Strep pyogenes or both
        - minor abrasions allow bacteria to enter the skin
            - they colonise and infiltrate the superficial layers
        - more common in children
            - bullous form mainly seen in under 2s, non-bullous in 2-5 year olds
            - adults may develop crusting or folliculitis around shaved areas (face, axillae)
        - rarely causes systemic upset
        - complications are rare, and mainly seen in neonates or immunosuppressed patients
        - these include:
            - meningitis
            - sepsis
            - secondary cellulitis
            - pneumonia
            - septic arthritis
            - post-streptococcal glomerulonephritis occasionally in young children
    - clinical classification
        - non-bullous impetigo

https://www.rcemlearning.co.uk/reference/cellulitis/#1568641615118-875f6c1f-9f70

            - initially red macules, then golden crusts
            - itchy but not painful
            - regional lymphadenopathy common
        - bullous impetigo

https://www.rcemlearning.co.uk/reference/cellulitis/#1568641615118-875f6c1f-9f70

- with fluid-filled lesions >0.5cm in diameter
- sloughing of the epidermis due to toxin production
- vesicles/bullae on face, buttocks, nappy area or trunk

- folliculitis
  - Staph aureus infection of hair follicles
- ecthyma

https://www.rcemlearning.co.uk/reference/cellulitis/#1568641615118-875f6c1f-9f70

- deeper, ulcerating
- associated with lymphadenitis

- impetiginous dermatitis
  - secondary infection of pre-existing skin disease or traumatised skin

- investigation
  - clinical diagnosis
  - consider swab for culture and sensitivities if:
    - suspected MRSA (swab from under crusts)
    - recurrent episodes (swab anterior nares and possibly axillae and perineum)
    - infected eczema (wet swab affected areas)
- management
  - topical antibiotics for localised areas
    - clean crust before applying

                - Fucidin is often ineffective due to resistance
                - mupirocin 2% is best choice
                - mupirocin nasal ointment should be used to eradicate nasal carriage when treating impetigo on the face
            - antibiotic creams are often combined with antiseptics such as chlorhexidine to combat resistance
            - systemic antibiotics for large areas/infected dermatitis
                - flucloxacillin
            - inpatient care for:
                - infants with bullous impetigo
                - patients with widespread impetiginized dermatitis who may develop sepsis/dehydration
                - neonates
                    - much higher incidence of sepsis/meningitis
            - follow up if lesions not cleared in 7 days
            - children should not return to school until lesions have cleared
            - carers should avoid contact with toys, towels etc. and wash clothing, toys and hands frequently
- erysipelas

https://dermnetnz.org/topics/erysipelas/

    - background
        - superficial form of cellulitis
            - involves the dermis and subcutaneous tissues
            - can be difficult to distinguish from cellulitis clinically
        - in erysipelas, the borders of the infection are sharply demarcated
        - appears as a fiery red rash
        - also known as St Anthony's fire
    - risk factors (cellulitis or erysipelas)
        - previous cellulitis/erysipelas
        - venous insufficiency
        - elderly
        - alcohol dependence
        - IV drug abuse
        - lymphoedema

        - overweight/obesity
        - athlete's foot/skin abrasions
        - inflammatory dermatoses
        - insect bites
        - pregnancy
    - causes
        - usually group A Strep
        - Strep pneumoniae
        - Klebsiella pneumoniae
        - Haemophilus influenzae type B
        - Yersinia enterocolitica
        - Moxarella spp
    - presentation
        - face or leg most commonly affected
            - arm or upper thigh next most common places
        - source of bacteria on the face is usually nasopharyngeal, may have had recent nasopharyngeal infection
        - may be recent skin trauma
        - malaise, chills and fever often precede skin lesion by up to 48 hours
            - may also have vomiting
        - sudden and rapid onset of skin infection with pruritus, burning and tenderness
            - lesions begin as small erythematous patch
            - progresses to fiery-red, indurated, tense shiny plaque
            - margins are raised, sharply demarcated and advancing
            - rapid enlargement over 3-6 days
            - local oedema, tenderness and warmth
            - skin can show streaking
            - may be regional lymphadenopathy
            - skin may then become deeper red with a bruise-like appearance and bright red leading edge
        - infection on the face is typically symmetrical
            - spreads from paranasal area to cheeks
            - tends to be unilateral elsewhere
        - systemic symptoms can continue once the infection is evident
        - severe infections can cause vesicles, bullae, petechiae and even necrosis
        - centre of erythema starts to clear in 7-10 days and returns to normal
        - desquamation and permanent pigmentary changes can occur
    - investigations
        - not normally required, diagnosis is clinical
        - culture of blister fluid may be needed if not resolving
    - management
        - as cellulitis
        - rest
        - elevation of limbs
        - analgesia (particularly NSAIDs)
        - NICE antibiotic recommendations:

                - flucloxacillin 500mg QDS in adults
                - erythromycin 500mg QDS (or clarithromycin or doxycycline if penicillin allergic
                - co-amoxiclav 625mg may be used for more severe infection or infection near the eyes/nose
                - if MRSA suspected, vancomycin, teicoplanin or linezolid can be added
                - duration is 7 days
                    - except patients with lymphoedema who require at least 14 days from when a clinical response is seen and may need several months
            - manage underlying conditions
            - emollient for skin hydration
            - marking the infection margins
            - follow up at 7 days and safety net for review at 48 hours if symptoms worsening
    - consider admission for:
        - severe and worsening infection or suspected necrotising fasciitis
        - systemic illness/vomiting
        - evidence of complications or suspected deep infection
        - facial infection
        - suspected orbital/periorbital cellulitis
        - immunocompromise
        - diabetes with unstable blood sugars
        - significant co-morbidity
        - lymphoedema
        - recurrent infection at the same site
        - child under 1
        - lack of home support/frailty/memory impairment
    - complications
        - uncommon
            - abscess formation
            - gangrene
            - thrombophlebitis/lymphangitis
            - chronic leg oedema
        - rare:
            - necrotising fasciitis
            - osteomyelitis
            - compartment syndrome
            - acute glomerulonephritis
            - endocarditis
            - septicaemia
            - Streptococcal toxic shock syndrome
    - prognosis
        - treatment without admission effective for over 90%
        - admission and IV antibiotics usually very successful otherwise
- cellulitis

- background
  - uncomplicated non-necrotising acute infection of the skin involving the hypodermis (mid-to lower dermis and subcutaneous tissue)
    - spares deeper structures such as fascia and muscle
- causes and risk factors same as erysipelas
- there is progression of erythema +/- fever for up to 72 hours after starting appropriate antibiotics
- erythema gradually resolves over weeks
  - discolouration can persist for months
- clinical features
  - acute onset
  - painful erythematous area of inflamed skin
    - not elevated
    - ill-defined demarcation of boundary
  - tender and warm on palpation
  - most common on the lower limbs
  - rarely bilateral
  - if more severe:
    - lymphangitis
    - lymphadenopathy
    - systemic features
- investigations
  - only required if systemically unwell
    - bloods and cultures
  - may require imaging for suspected foreign body (x-ray) or gas in tissue (US)
- management
  - as for erysipelas
- classification
  - class I
    - no systemic toxicity
    - no uncontrolled co-morbidity
  - class II
    - systemically ill OR co-morbidity complicating infection
  - class III
    - signs of marked systemic illness (confusion, tachycardia, hypotension) or severe co-morbidity
  - class IV
    - sepsis or life-threatening infection such as necrotising fasciitis
- admission required:
  - class IV cellulitis
  - class III cellulitis
  - severe or rapidly deteriorating cellulitis
  - under 1 year or frail
  - immunocompromised
  - significant lymphoedema
  - facial cellulitis (unless very mild)
  - suspected orbital or preorbital cellulitis

        - class II cellulitis if community support unavailable (e.g. monitoring, OPAT)
        - symptoms and signs suggesting a more severe illness such as osteomyelitis or septic arthritis
- differentials for cellulitis
    - panniculitis

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        - inflammation of the subcutaneous fat +/- associated vasculitis
        - affected skin feels thickened and woody
        - there may be red or darker, brownish discolouration
        - often tender
        - affected areas usually appears as nodules or lumps under the skin
            - may be a plaque of thickened skin
        - there may be purpuric discolouration, bullae and erosions
        - causes include:
            - collagen vascular disorders
                - rheumatoid arthritis
                - sarcoidosis
                - polyarteritis nodosum
            - Crohn's disease
            - steroids
            - necrobiosis lipoidica
            - erythema nodosum
    - post-phlebitic limb

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- thrombophlebitis results in chronic skin discolouration and scarring, sometimes with oedema
- no erythema, generally no discomfort unless there is associated eczema

- leg eczema

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- discoid eczema can occur at any age
  - results in round or oval plaques
  - dry discoid eczema is often relatively non-itchy and often due to over dry skin
  - exudative/wet discoid eczema is often triggered by an injury to the skin colonised by Staph aureus
  - starts with a single patch on one leg
    - multiple lesions soon appear on both legs and may affect the trunk and arms
- atopic eczema is mainly seen in children
  - commonly found behind the knees but can affect other areas during flares or if secondarily infected
  - characteristically very itchy

- venous insufficiency

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  - also called venous eczema, stasis eczema, gravitational eczema
  - secondary to poor venous drainage in the leg
  - episodes of cellulitis or deep vein thrombosis damage the valves, causing back pressure to develop and fluid to collect in the tissues, causing chronic skin inflammation
  - leg usually swollen due to inflammation and lymphoedema
  - dermatitis is usually bilateral with yellow crusting, scaling and marked itching
  - may become secondarily infected
    - increased discomfort and erythema help to differentiate acute cellulitis from the chronic picture
- thrombophlebitis

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  - inflammation of the superficial leg veins
  - tender red or purple subcutaneous cord and associated swelling along the track of the vein
  - may be secondary to deep vein insufficiency, cannulation or injection or malignancy (migratory thrombophlebitis, Trousseau syndrome)
- deep vein thrombosis

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        - does not normally cause significant erythema or blistering
        - not generally tender but can have tenderness along the deep veins
        - pitting oedema may be present, with swelling >3cm larger than the unaffected side
- dermatophytosis
    - background
        - fungal infections caused by dermatophytes, a group of fungi that invade and grow in dead keratin
        - tend to grow outwards on skin, producing ring-like patterns
        - treatment is usually successful but depends on site of infection and compliance with treatment
    - pathophysiology
        - infection limited to dead layers of skin
            - encouraged by warm, damp local environment
        - transmitted by anthropophilic, geophilic or zoophilic spread
        - most common organisms:
            - Trichophytons rubrum
            - Trichophytons tonsurans
            - Trichophytons interdigitale
            - Trichophytons mentagrophytes
            - Microsporum canis
            - Epidermophyton floccosum
    - clinical classification
        - scalp – tinea capitis

https://dermnetnz.org/topics/tinea-capitis/

- feet – tinea pedis

https://dermnetnz.org/topics/tinea-pedis/

  - hands – tinea manuum
  - nails – tinea unguium or onychomycosis

https://dermnetnz.org/topics/fungal-nail-infections/

  - beard area – tinea barbae
  - groin – tinea cruris
  - body – tinea corporis

https://dermnetnz.org/topics/tinea-corporis/

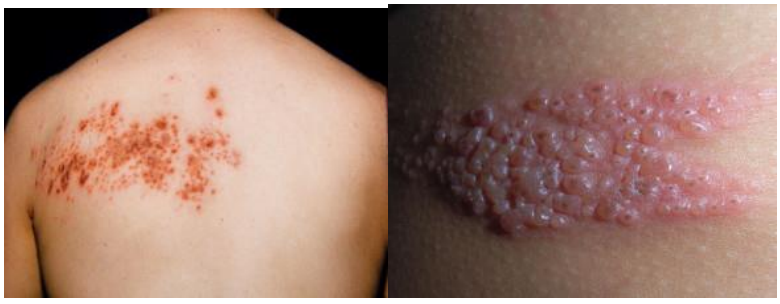
- history
  - itch, rash, nail discolouration
  - hair loss with tinea capitis
  - symptoms of secondary infection (cellulitis, impetigo)
  - history of playing contact sports
  - immunocompromise
- examination

        - tinea pedis
            - web of toes may be macerated and erythematous
            - commonly plantar surface
            - may be erythema, vesicles, pustules
        - tinea capitis
            - hair loss with broken hairs at the surface
            - variable clinical appearance including scaly, crusting pustules and/or black dot alopecia
        - tinea unguium
            - onycholysis – separation or the nail from the nail bed – is common
            - nail dystrophy with thickening and discolouration
        - tinea corporis
            - annular scaly plaques with raised edges
            - may be vesicles and pustules
            - lesions typically on exposed areas
        - tinea manuum
            - usually alongside tinea pedis
            - typically one hand
            - scaling and redness
            - incorrect diagnosis and steroid use may exacerbate condition
        - tinea cruris
            - usually in men
            - erythematous with central clearing and raised edge
    - investigation
        - microscopy of skin and nail specimens for hyphae and spores
        - fungal culture can identify the species but not reliable and can take 6 weeks
        - Wood's light (ultraviolet) is useful for tinea capitis which produces fluorescence
            - tinea corporis and tinea cruris do not produce this
    - management
        - hygiene measures
            - keep affected skin cool and dry
            - wear cotton, absorbent clothing
            - avoiding scratching
            - dry thoroughly after washing
            - do not share towels, wash frequently
            - cover feet in communal pools, changing areas and gyms if feet affected
        - antifungals
            - nails
                - topical amorolfine nail lacquer for 6-12 months for mild infections
                - oral terbinafine or itraconazole
            - skin
                - topical antifungals (imidazoles)
                - systemic treatment only in severe, extensive or systemic infection

        - scalp
            - oral griseofulvin or terbinafine alongside topical shampoo
- viral skin infection
    - herpes simplex

https://dermnetnz.org/topics/herpes-simplex/

        - type 1 or 2 HSV
        - remains latent in spinal cord dorsal root nerves supplying sensation to the skin
        - can inoculate to new areas during an attack
        - mainly occurs in infants and young children
        - transmitted by direct or indirect contact with someone with active herpes simplex
            - it is infectious for 7-12 days
        - minor injury to the skin helps inoculation to occur
        - complications include:
            - eye infection with corneal ulceration (dendritic)
            - throat infection
            - eczema herpeticum
            - erythema multiforme
            - cranial/facial nerve involvement
            - disseminated infection in immunosuppressed patients
        - treatment not required for mild eruptions
            - acyclovir 200mg 5 times/day for 5 days or valaciclovir 1g TDS for 7 days if needed
    - herpes zoster



https://dermnetnz.org/topics/herpes-zoster/

        - localised, blistering, painful rash caused by reactivation of varicella zoster virus
        - dermatomal distribution

                - cutaneous distribution of one or two adjacent sensory nerves
                - usually unilateral with sharp cutoff
            - more common in adults, cancer patients, immunosuppressed patients
            - pain usually precedes rash
                - may be severe
                - may be in one place or spread out
                - patient may feel unwell with fever and headache
                - may have local tender lymphadenopathy
            - rash starts as red papules with new lesions erupting for several days
                - they blister or become pustular then crust over
            - most common locations are chest, neck, forehead and lumbo-sacral areas
            - recovery is usually in 2-3 weeks in children, 3-4 weeks for adults
            - complications
                - involvement of several dermatomes
                - eye complications
                - deep blisters destroying the skin and leaving scars
                - muscle weakness such as facial nerve palsy (Ramsay Hunt syndrome)
                    - 50% chance of complete recovery
                - infection of internal organs/CNS
                - post-herpetic neuralgia
                    - pain lasting more than a month after onset
                    - overlying skin is numb or hypersensitive
                    - management options include:
                        - early antivirals
                        - local anaesthetic applications
                        - topical capsaicin
                        - tricyclic antidepressants (e.g. amitriptyline)
                        - anti-epileptics (e.g. gabapentin, pregabalin)
                        - transcutaneous electrical nerve stimulation or acupuncture
                        - botulinum toxin to affected area
            - management
                - antivirals if started 1-3 days after onset
                - acyclovir 800mg 5 times/day for 7 days
            - patients are infectious to those who have not had chickenpox
    - molluscum contagiosum

https://dermnetnz.org/topics/molluscum-contagiosum/

- caused by a DNA pox virus
- common, mostly in children
- can be spread by direct contact, contaminated objects, sexual contact
- incubation period 2-8 weeks
- assumed to be infectious whilst visible lesions present
    - mean duration of lesions is 8 months
- firm, smooth, umbilicated papules usually 2-5mm in diameter
    - can be skin coloured, white or slightly yellow
- usually on trunk or extremities in children, lower abdomen, inner thighs or genital region in adults
- may be single or in clusters of up to 30 or more
- most cases clear up spontaneously within 18 months
- avoid scratching to reduce risk of infection/scarring

- warts
    - common and usually harmless
    - caused by human papillomavirus (HPV)
    - majority resolve spontaneously
    - acquired by direct contact or environmental contact
    - trauma and wetness contribute to contraction
    - classified according to appearance or site
        - common wart (verruca vulgaris)
            - papules and nodules with a keratotic and papillomatous surface
            - occur anywhere but common on hands
        - plane wart or flat wart (verruca plana)
            - slightly elevated, flat-topped
            - may be single or in a group
        - plantar wart or verruca (verruca planis)
            - warts on the sole of the foot
        - genital wart (condyloma acuminatum)
        - periungual wart
            - more common in people who bite their nails
        - filiform wart (verruca filiformis
        - finger-like warts of hyperkeratotic projections
        - most often on face or neck

- management
    - eventually will resolve without therapy
    - salicylic acid has the best evidence base
        - warts should be pared down first
        - daily treatment for at least 12 weeks
    - cryotherapy
        - with liquid nitrogen every two weeks until wart is gone (up to four months)

- orf

https://dermnetnz.org/topics/orf/

- contracted from sheep and goats
- caused by a parapox virus
- occurs in farmers, butchers, children who bottle-feed lambs or play in pastures
- incubation period 5-6 days
- lesions usually solitary
    - small, firm, red or reddish-blue
    - form a lump that enlarges to form a flat-topped, blood-tinged pustule or blister
    - fully developed lesion is usually 2-3cm in diameter, may be as large as 5cm
    - there appears to be pus underneath but incising reveals firm red tissue
    - lesion is sometimes irritable during early stages and is often tender
- usually occur on fingers, hands or forearms but can be on the face
- may have lymph lines tracking on medial side of elbow up to axilla
- may be mild fever
- allergy to the virus may cause erythema nodosum 10-14 days later
- lesion can be covered to prevent spread (human to human transmission is rare)
- resolves spontaneously in 3-6 weeks

- hand, foot and mouth disease

https://dermnetnz.org/topics/hand-foot-and-mouth-disease/

- mild short-lasting viral infection
- most often affects young children
- characterised by blisters on hands, feet and in the mouth
- caused by an enterovirus, most commonly Coxsackie virus A16
- very infectious, so members of a family or school class may be affected
- lesions:
    - dorsal and palmar surfaces of hands and feet
    - progress from flat pink patches to small elongated greyish blisters and these peel off within a week leaving no scars
    - small vesicles and ulcers in and around the mouth, palate and pharynx
        - sometimes painful and prevent child from eating
    - red macules and papules on the buttocks and sometimes arms
    - lesions can occur on the genitalia
- can present atypically with more widespread, severe rash, larger blisters and subsequent skin peeling and/or nail shedding
- does not require any specific treatment, just supportive care
- children do not need to be kept off school as it is a mild illness
    - blisters remain infective until dried up (usually within a few days)
    - stools are infective for up to a month – thorough handwashing helps prevent spread

DC6 necrotising fasciitis

https://www.rcemlearning.co.uk/reference/cellulitis/#1568641615118-875f6c1f-9f70

- background
    - severe bacterial soft tissue infection

    - marked by oedema and necrosis of subcutaneous tissues with involvement of the adjacent fascia and painful red swollen skin over affected areas
    - may resemble cellulitis but is rapidly progressive
- classification
    - type I
        - polymicrobial
        - obligate and facultative anaerobes
        - affects trunk and perineum
        - associated with diabetes
    - type II
        - monomicrobial
        - beta-haemolytic group A Streptococcus
        - affects limbs
    - type III
        - Clostridium spp, Gram-negative bacteria, Vibrios spp, Aeromonas hydrophilia
        - affects limbs, trunk and perineum
        - associated with trauma (and seafood consumption for Aeromonas)
    - type IV
        - Candida spp
        - affects limbs, trunk, perineum
        - associated with immunosuppression
- risk factors
    - diabetes
    - alcohol abuse
    - peripheral vascular disease
    - renal failure
    - odontogenic infection
    - malignancy
    - chicken pox
    - local penetrating trauma or animal bite
    - recent surgery (e.g. abdominal, peritoneal)
- clinical assessment
    - history
        - spreading erythema
        - sepsis (fever, chills, myalgia)
        - severe constant pain out of proportion to clinical findings
        - cutaneous anaesthesia
        - evidence of developing end organ failure
        - underlying risk factors
        - commonly at an extremity but can affect anywhere
    - examination
        - local
            - erythematous, tender, swollen
            - skin becomes smooth, shiny and tensely swollen
                - darkens and becomes patchy with blisters and bullae
            - wooden-hard feel to subcutaneous tissue

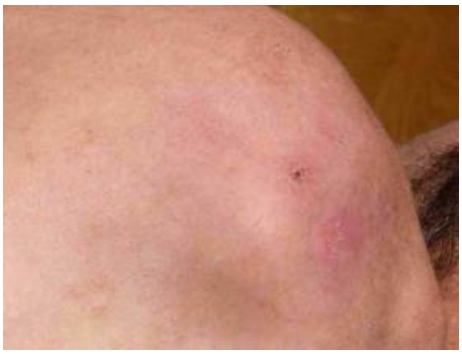
                - oedema beyond the margin of erythema
                - crepitus (gas gangrene)
                - rapid spread on re-examination
            - systemic
                - fever, tachycardia
                - haemodynamic instability
- investigations (only if they do not delay surgical intervention)
    - blood cultures
    - FBC
    - CRP
    - coagulation profile (risk of DIC)
    - U&Es (risk of renal failure)
    - CK
    - imaging:
        - x-ray
            - gas in subcutaneous tissue
        - CT
            - subcutaneous air
        - MRI
            - shows extent of fascial necrosis
            - can guide limits of debridement
- LRINEC score (laboratory indicators of necrotising fasciitis)
    - shown to have poor sensitivity and specificity
    - not clinically validated and should not replace clinical judgment
    - score of 6 or more indicates high suspicion
    - elements:
        - CRP
            - <150 (0)
            - >150 (4)
        - WCC
            - <15 (0)
            - 15-25 (1)
            - >25 (2)
        - Hb
            - >135 (0)
            - 110-135 (1)
            - <110 (2)
        - urea
            - <15 (0)
            - >15 (1)
        - sodium
            - ≥135 (0)
            - <135 (1)
        - creatinine
            - ≤141 (0)
            - >142 (2)
        - glucose

                - ≤10 (0)
                - >10 (1)
- management
    - aggressive management of septic shock
    - extensive urgent surgical debridement
    - antimicrobial therapy (broad spectrum)
        - empiric – meropenem and clindamycin
    - seek and treat underlying cause/co-morbidities
    - early involvement of surgeons and intensive care
- prognosis
    - mortality 30-40% with appropriate therapy
    - increased mortality in co-morbid patients
    - mortality directly proportional to delay in diagnosis and treatment

DC7 pressure ulcers

- background
    - localised injury to the skin and/or underlying tissue
        - usually over a bony prominence
        - result of pressure or pressure in combination with shear
    - have a number of associated factors
    - preventable
- pathophysiology
    - predisposing factors (4Ps)
        - pressure +/- shear
        - poor perfusion
        - poor healing
        - poor 'padding'/prominences
    - development of ulcers
        - can develop within 2-6 hours
        - most vulnerable areas:
            - coccyx
            - heels
            - sacrum
            - femoral trochanter
        - skin and subcutaneous tissues compressed or subjected to shear forces
            - results in decreased perfusion and tissue necrosis
- risk factors
    - patient and care
        - advanced age
        - male
        - white race
        - smoker
        - low BMI
        - impaired mobility
        - urinary and faecal incontinence
        - history of pressure ulcers
        - altered mental state

        - fever
        - hypotension
        - requiring physical restraints
        - inadequate care
    - co-morbidities
        - malignancy
        - diabetes
        - stroke
        - pneumonia
        - heart failure
        - sepsis
        - malnutrition
        - renal failure
    - laboratory
        - anaemia
        - lymphopaenia
        - hypoalbuminaemia
- assessment
    - risk scoring
        - Waterlow, Braden or Norton score
    - assess ulcer features and possible causes
        - location, area, depth, drainage, tissue type, presence of cellulitis
        - ulcer staging
        - underlying risk factors and reversible conditions
    - staging
        - International NPUAP-EPUAP
        - stage I



https://dermnetnz.org/topics/pressure-ulcer/

            - non blanching erythema
            - may look like mild sunburn
            - skin may be tender, itchy, painful
        - stage II – partial thickness
            - skin is red, swollen, painful
            - blisters may be present
            - upper layers of skin begin to die
        - stage III – full thickness skin loss involving subcutaneous tissue (fascia intact)
            - crater like ulceration

        - wound prone to infection
    - stage IV – full thickness tissue loss involving underlying bone, tendon, muscle or cartilage

https://dermnetnz.org/topics/pressure-ulcer/

        - blackened dead tissue (eschar) may be seen in deep open wounds
- management
    - prevention
        - risk assessment and monitoring
        - treat underlying critical illness and reversible factors
        - mobilise
        - manage urinary incontinence and diarrhoea
        - avoid pressure and friction
            - e.g. 2 hourly repositioning
            - appropriate padding and mattresses
        - adequate nutrition
        - skin care – keep clean and dry
        - minimise sedation
        - promote wound healing
            - stop medications that impair wound healing
            - control diabetes
            - optimise local and systemic perfusion
        - staff education
    - specific therapy
        - debridement
            - mechanical (wet to dry gauze)
            - autolytic (dressings that promote breakdown of necrotic tissues by the body's enzymes)
            - enzymatic (proteolytic enzymes)
            - scalpel or laser debridement
            - maggot therapy
        - managing bacterial burden
            - silver impregnated dressing, silver sulfadiazine
        - exudate management
            - avoid excessive moisture
        - monitor healing
        - surgery (rarely required)
            - direct closure

                - skin graft
                - skin flaps
                - musculocutaneous flaps
                - free flaps
                - stents and revascularisation
            - adjunctive therapies
                - electrical stimulation
                - topical growth factors
                - skin equivalents
                - hyperbaric oxygen

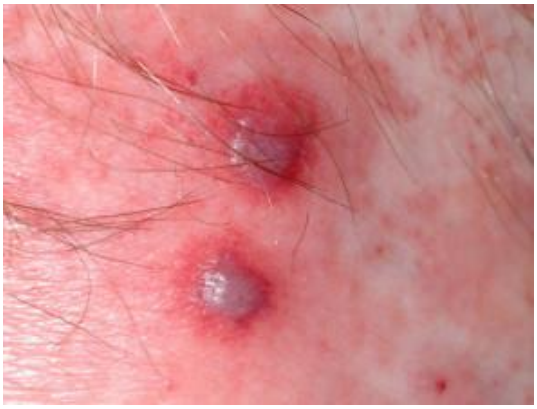
DC8 purpuric rash including Henoch Schönlein purpura

- background
    - purpura is a purplish discolouration of the skin produced by small bleeding vessels near the surface
    - may occur in the mucous membranes (mouth and internal organs)
    - indicative of an underlying cause of bleeding
    - called petechiae if <1cm in diameter
    - larger, deeper purpura are referred to as ecchymoses or bruising
    - may occur with normal platelet counts (non-thrombocytopaenic purpura) or decreased platelet counts (thrombocytopaenic purpura)
        - usually purpura are indicative of a problem with the platelet system rather than the clotting system but this must also be considered
- examination
    - rash that does not blanch on pressure
    - note nature of lesions
        - size
        - confluence
        - associated blisters (and what they contain: blood, exudate, pus)
    - note location of lesions
    - look for mucous membrane lesions
    - tenderness may suggest an inflammatory process
    - hepato-splenomegaly
- history
    - age of patient
        - HSP tends to occur in children
        - senile purpura is confined to the elderly
        - leukaemia and myeloproliferative disorders can occur at any age
    - how long rash has been present and whether it is changing
        - meningococcal septicaemia rash will be very recent and changing rapidly
    - establish if patient is otherwise well
    - whether there has been general easy bruising
    - recent travel history
- causes
    - non-thrombocytopaenic purpura
        - congenital

                - hereditary haemorrhagic telangiectasia (Osler-Weber-Rendu syndrome)
                - connective tissue disorders (e.g. Ehlers-Danlos, pseudoxanthoma elasticum)
                - congenital CMV
                - congenital rubella
            - acquired
                - severe infection
                    - e.g. septicaemia, meningococcal infections, measles
            - allergic
                - Henoch-Schönlein purpura
                - connective tissue disorders (SLE, rheumatoid arthritis)
            - drug induced
                - e.g. steroids, sulphonamides
            - others
                - senile purpura
                - trauma
                - scurvy
                - dependent purpura with venous hypertension
                - facitial purpura
    - thrombocytopaenic purpura
        - impaired platelet production
            - generalised bone marrow failure
                - e.g. leukaemia, aplastic anaemia, myeloma, marrow infiltration by solid tumours
            - selective reduction in megakaryocytes
                - e.g. co-trimoxazole, chemicals, viral infections
        - excessive platelet destruction
            - immune problems
                - immune thrombocytopaenia
                - secondary immune thrombocytopaenia (SLE, viral infections, drugs)
            - coagulation problems
                - e.g. disseminated intravascular coagulation (DIC)
                    - massive ecchymosis with sharp, irregular borders of deep purple colour and an erythematous halo
                    - can evolve to haemorrhagic bullae and blue-black gangrene
                - haemolytic uraemic syndrome
        - sequestration of platelets
            - e.g. splenomegaly
        - dilutional loss
            - e.g. post massive transfusion of stored blood
- investigations (guided by differential)
    - FBC including platelets
    - LFTs
    - coagulation screen

- plasma electrophoresis
    - may show hypergammaglobulinaemia, paraproteinaemia, cryoglobulinaemia
- autoantibody screen for connective tissue disorders

- Henoch-Schönlein purpura



https://dermnetnz.org/topics/henoch-schoenlein-purpura/

- background
    - IgA vasculitis (small vessel immune complex)
    - most common childhood vasculitis
    - uncertain underlying pathogenesis
        - structural end organ injury is caused by deposition of IgA molecules due to a reduction in glycosylation
- history
    - preceding infection affecting GI system or upper respiratory tract
- clinical manifestations (tetrad)
    - purpura
        - most often on areas of pressure or gravitational dependence
    - arthritis or arthralgia (50-75%)
    - abdominal pain (50%)
        - often due to small bowel involvement
        - can be complicated by intussusception
    - renal involvement (25-50%)
        - most common long term consequence of IgA vasculitis
        - can initially present with hypertension or evidence of nephrotic syndrome
- examination
    - hypertension
        - BP must be measured at presentation and follow up
    - skin
        - palpable purpura, petechiae and ecchymoses
            - usually symmetrical
            - on gravity/pressure dependent areas
        - may be preceded by urticarial, erythematous, maculopapular or bullous skin lesions
        - painful non-pitting subcutaneous oedema

        - commonly peri-orbital (in non-ambulant child) and/or dependent areas (hands, feet, scrotum)
    - joints
        - arthralgia +/- arthritis
            - usually affects large joints of lower limbs, rarely upper limbs
            - usually no significant effusion or warmth
    - abdominal
        - diffuse abdominal pain
        - generalised tenderness
        - signs of bowel obstruction
        - peritonism
        - most common complication is intussusception, others include GI haemorrhage, bowel ischaemia/necrosis/perforation, protein-losing enteropathy, pancreatitis
    - genital
        - testicular pain +/- orchitis +/- necrosis
            - need to exclude testicular torsion
        - cord haematoma
    - respiratory (rare)
        - respiratory distress
            - diffuse alveolar haemorrhage
    - neurological (rare)
        - changes in mental status
            - labile mood, apathy, hyperactivity, encephalopathy
        - focal neurological signs
            - consider intracranial haemorrhage
    - renal
        - haematuria
            - microscopic or macroscopic
        - proteinuria
            - mild to severe
            - nephrotic syndrome
            - acute nephritic syndrome
        - hypertension
        - renal impairment/renal failure
- investigations
    - urinalysis
        - usually the only investigation required
        - if there is hypertension, macroscopic haematuria or significant proteinuria:
            - formal urine microscopy and urinary protein-creatinine ratio
            - U&Es and albumin
    - additional investigations if diagnosis unclear (e.g. differentials of ITP, leukaemia, meningococcal infection) or to identify potential complications
        - FBC, U&E, albumin
        - blood and urine cultures
        - abdominal imaging

- autoimmune screen

- management
  - if normal BP or microscopic haematuria only:
    - home with advice and follow up if no red flags:
      - looks unwell
      - pain poorly controlled with simple analgesia
      - testicular involvement
      - peritonism
      - respiratory or neurological features
  - if red flags, abnormal blood pressure or abnormal urinalysis:
    - additional investigations
    - discuss with paediatric team +/- renal specialist
    - steroids for abdominal/joint pain
    - admission
- follow up
  - repeat urinalysis and BP
    - weekly for first month
    - fortnightly from weeks 5-12
    - single reviews at 6 and 12 months
  - return to weekly reviews if clinical disease flare or additional signs of renal involvement
  - no further follow up after 12 months if no significant renal involvement and normal urinalysis
  - pain
    - mild
      - rest and elevation for subcutaneous oedema
      - regular paracetamol and short course of NSAIDs if not contraindicated
    - moderate to severe
      - consider steroids (may reduce duration of symptoms
        - steroids do not impact the rate of long term renal complications
        - oral prednisolone should be 1-2mg/kg/day (up to 60mg) whilst symptoms persist followed by weaning
  - admission under paeds
    - serious abdominal complications
    - severe debilitating pain
    - severe renal involvement
    - neurological or pulmonary involvement
    - if steroids considered
    - development of hypertension, proteinuria, macroscopic haematuria
  - consideration for renal specialist referral
    - hypertension
    - abnormal renal function
    - macroscopic haematuria for 5 days
    - nephrotic syndrome
    - acute nephritic syndrome

        - persistent proteinuria
    - prognosis
        - good
        - first episode usually resolves within 4 weeks if no significant renal involvement
            - rash is often the last symptom to clear
        - joint pain usually resolves within 72 hours
        - uncomplicated abdominal pain usually resolves within 24-48 hours
        - in 25-35% of patients, HSP recurs at least once within 4 months of the initial presentation
            - subsequent episodes are usually milder and shorter in duration
        - 90% of those who develop renal complications do so within 2 months of onset, 97% within 6 months

DC9 Stevens-Johnson syndrome

https://dermnetnz.org/topics/stevens-johnson-syndrome-toxic-epidermal-necrolysis/

- background
    - immune complex mediated hypersensitivity disorder
    - ranges from mild skin and mucous membrane lesions to a severe, sometimes fatal, systemic illness
    - overlaps with toxic epidermal necrolysis
    - incidence estimated at 2-3 per million population per year in Europe
    - much more common in patients with HIV
    - more common in females
    - most patients are aged 10-30
- causes
    - 75% caused by medications, 25% by infections and other causes
    - medications:
        - allopurinol
        - carbamazepine
        - sulphonamides
        - antiviral agents
            - nevirapine

                - abacavir
            - anticonvulsants
                - phenobarbital
                - phenytoin
                - valproic acid
                - lamotrigine
            - imidazole antifungals
            - NSAIDs (oxicam type)
            - salicylates
            - sertraline
            - bupropion
        - infection
            - viral
                - herpes simplex
                - Epstein Barr
                - enteroviruses
                - HIV
                - coxsackievirus
                - influenza
                - hepatitis
                - mumps
                - lymphogranuloma venereum
                - rickettsia
                - variola
            - bacterial
                - group A beta haemolytic streptococcus
                - diphtheria
                - brucellosis
                - mycobacteria
                - Mycoplasma pneumoniae
                - tularaemia
                - typhoid
            - fungal
                - coccidioidomycosis
                - dermatophytosis
                - histoplasmosis
            - protozoal
                - malaria
                - trichomoniasis
        - immunisation
            - e.g. measles, hepatitis B
- symptoms
    - non-specific upper respiratory tract infection
        - fever
        - sore throat
        - chills
        - headache

        - arthralgia
        - vomiting and diarrhoea
        - malaise
    - mucocutaneous lesions
        - develop suddenly
        - clusters last from 2-4 weeks
        - lesions are not usually pruritic
    - severe oromucosal ulceration
    - may have cough productive of thick purulent sputum
    - may have dysuria or inability to pass urine if genitourinary involvement
    - ocular symptoms:
        - painful red eye
        - purulent conjunctivitis
        - photophobia
        - blepharitis
- signs
    - general examination
        - fever
        - tachycardia
        - hypotension
        - altered level of consciousness
        - seizures
        - coma
    - skin
        - lesions anywhere, but most commonly palms, soles, dorsum of hands and extensor surfaces
        - rash may be confined to any body area, most commonly the trunk
        - rash can begin as macules that develop into, papules, vesicles, bullae, urticarial plaques, confluent erythema
        - centre of lesions may be vesicular, purpuric or necrotic
        - typical lesion has a target appearance
        - lesions may become bullous and rupture
            - skin is susceptible to secondary infection
        - urticarial lesions are not normally pruritic
        - mechanical pressure to skin leads to blistering within minutes or hours (Nikolsky sign)
    - muscle involvement
        - erythema
        - oedema
        - sloughing
        - blistering
        - ulceration
        - necrolysis
    - eye
        - conjunctivitis
        - corneal ulceration
    - genital
        - erosive vulvovaginitis or balanitis

- investigations
    - U&Es, glucose and bicarbonate to assess severity and level of dehydration
    - diagnosis is based on clinical classification and histopathology
- management
    - acute phase
        - identify and remove causative drug or underlying cause
        - ALDEN (Algorithm for assessment of Drug-induced Epidermal Necrolysis) may be useful
        - rapid assessment of prognosis using SCORTEN score (SCOre for Toxic Epidermal Necrolysis)
            - illness severity score to predict mortality in SJS and TEN
            - one point for each of seven criteria present at time of admission:
                - age >40
                - presence of malignancy
                - heart rate >120
                - initial percentage of epidermal detachment >10%
                - serum bicarbonate <20 mmol/L
                - serum urea >10 mmol/L
                - serum glucose >14 mmol/L
            - patients with a score >3 should be managed in intensive care
    - supportive care
        - attention to airway and haemodynamic stability
        - IV fluid and electrolyte replacement if severe fluid loss
        - pain control
        - treat lesions in the same way as burns
        - mouth: mouthwashes and topical anaesthetics
            - reduces pain and improves oral intake
        - eyes: frequent ophthalmology assessment, frequent eye drops (may require antibiotic and steroid)
    - treat secondary infections
    - immunomodulation may be attempted but evidence is lacking
        - pulsed systemic steroids may help to balance dampening of immune system with poor wound healing
- complications
    - dehydration and acute malnutrition
    - shock and multiorgan failure
    - thromboembolism and DIC
    - gastrointestinal ulceration, necrolysis, stricture, perforation
    - secondary infection and scarring of skin
    - mucosal pseudomembrane formation
        - can lead to mucosal scarring and loss of function in the organ system involved
    - respiratory failure
        - caused by mucosal shedding in the tracheobronchial tree
    - eye complications
        - corneal ulceration
        - anterior uveitis

        - sight impairment secondary to severe keratitis or panophthalmitis in 3-10% of patients
    - vaginal stenosis and penile scarring
    - renal complications
        - uncommon
        - renal tubular necrolysis and acute kidney injury may occur
- prognosis
    - mortality rate up to 10% for SJS and at least 30% for TEN
        - mortality rate correlates with SCORTEN score
            - mortality >90% for score of 5 or more
            - mainly from systemic infection and multiple organ failure
- sequalae
    - occur in more than 50% of TEN survivors and many SJS survivors
        - skin
            - hyperhidrosis
            - xeroderma
            - reversible hair loss
            - heat and cold sensitivity
            - scarring
            - irregular pigmentation
        - nail dystrophy
        - mucous membranes
            - vaginal, urethral and anal strictures
            - persistent mucosal erosion
        - ocular
            - xerophthalmia
            - photophobia
            - symblepharon (adhesions between palpebral and bulbar conjunctiva)
            - synechiae (adhesions of the iris to ocular structures)
            - entropion (lid turned inward)
            - meibomian gland dysfunction
            - sight impairment

DC10 toxic epidermal necrolysis

https://dermnetnz.org/topics/stevens-johnson-syndrome-toxic-epidermal-necrolysis/

- background
    - acute-onset, potentially life threatening, idiosyncratic mucocutaneous reaction

    - usually related to a new medication
    - involves widespread full-thickness epidermal necrosis
        - causes erythema and sloughing of the skin and mucosa
        - involves internal and external surfaces
        - skin looks similar to a scald
    - usually affects trunk, face and one or more mucous membranes
    - likely to be a more severe variant of Stevens-Johnson Syndrome
- causes
    - thought to be an immune-complex-mediated hypersensitivity reaction to the presence of toxic drug metabolites accumulating in the skin
        - results in the destruction of keratinocytes caused by cytotoxic T lymphocytes
- risk factors
    - drugs – usually within a few days to two months after starting
        - more than 200 drugs associated with TEN
        - most common drugs:
            - sulphonamides
            - ampicillin
            - quinolones
            - cephalosporins
            - anticonvulsants
                - phenobarbital
                - phenytoin
                - carbamazepine
                - lamotrigine
                - valproate
            - allopurinol
            - antiretrovirals
            - corticosteroids
            - NSAIDs (especially 'oxicam')
    - immunisations
    - bone marrow or organ transplantation
    - infections
        - e.g. mycoplasma, HIV
    - systemic lupus erythematosus
    - malignancy
    - idiopathic
    - likely to be a genetic component
- epidemiology
    - 1-2 cases per million population per year worldwide
    - more common in elderly people
        - possibly due to polypharmacy
        - can occur in any age group
- presentation
    - prodromal phase
        - lasts 2-3 days
        - fever

        - upper respiratory tract infection symptoms
        - conjunctivitis
        - pruritus
        - malaise
        - arthralgia
        - myalgia
    - mucous membrane involvement
        - occurs early in 90% of cases
        - commonly precedes other symptoms
        - can involve:
            - conjunctivae
            - buccal mucosa
            - nasal mucosa
            - pharyngeal mucosa
            - tracheobronchial mucosa
            - perineal mucosa
            - vaginal mucosa
            - urethral mucosa
            - anal mucosa
    - rash
        - ill-defined red 'burning/painful' macular rash
            - spreads from face or upper trunk
            - bullae form and coalesce
                - generally increase in number over 3-4 days (or sometimes hours)
            - epidermis can slough in sheets
    - hyperpyrexia may occur
    - hypotension and tachycardia
        - secondary to dehydration and hypovolaemia
    - positive Nikolsky's sign
        - areas of seemingly normal skin, when rubbed, have the epidermis easily separate from its underlying surface
- differential
    - staphylococcal scalded skin syndrome
    - burns
    - bullous impetigo
    - pemphigus, pemphigoid
        - usually slower onset
    - epidermolysis bullosa hereditaria
    - SLE
    - scarlet fever
        - has desquamation but no bullae
    - other drug eruptions
    - erythema multiforme
    - bullous lichen planus
    - toxic shock syndrome
        - toxin-mediated reaction to staphylococcal infection

    - erythroderma/exfoliative dermatitis
- investigations
    - no confirmatory test
    - skin biopsy and immunofluorescence staining may be used
        - full thickness epidermal necrosis plus epidermal detachment and sloughing
    - percentage of body area affected distinguishes SJS from TEN
        - <10% BSA – SJS
        - 10-30% BSA – SJS/TEN overlap syndrome
        - >30% BSA - TEN
    - monitor FBC, U&E, albumin, total protein and proteinuria
    - blood, urine and skin cultures
- management
    - early transfer to ITU/burns unit
        - A: may need intubation due to mucosal involvement
        - B: protective lung ventilation strategy
            - can develop secretions and sloughing
        - C: fluid resuscitation similar to in a burns patient
            - large volumes proportional to BSA
            - aim for urine output >1 ml/kg/hr
        - D: multimodal analgesia – may required sedation, intubation and ventilation for analgesia
        - E: keep warm, isolate to reduce risk of secondary infection, humidified environment
    - specific treatment
        - stop offending agent
        - identify and treat underlying disease and secondary infection
        - burns dressings
        - avoid antibiotics that may exacerbate condition (e.g. sulphas)
        - IgG and steroids are controversial
        - consider plasma exchange
        - debridement of necrotic areas of skin may be needed, with skin grafts to protect exposed dermis
            - prevent fluid and protein loss
            - prevent infection
            - control pain
        - oral hygiene and ophthalmological treatment
- complications
    - acute
        - widespread skin infection and septicaemia
        - pneumonia and respiratory failure
        - dehydration
            - increased fluid loss
            - inability to take oral fluids if mouth involved
        - hypovolaemic shock
        - multi-organ failure
        - thromboembolism and DIC
        - thermoregulatory disturbance

- long term
    - ocular complications (in 50%), may lead to blindness
        - dry or watery eyes (46%)
        - conjunctivitis
        - corneal ulcers
        - symblepharon
        - ectropion and entropion
        - trichiasis
        - synechiae
    - joint contractures
    - loss of nails
    - stomatitis and mucositis
    - gastrointestinal haemorrhage
    - oesophageal strictures and dysphagia
    - phimosis
    - gynaecological and obstetric complications
        - premature labour
        - long-term painful genitourinary lesions
        - vaginal stenosis
        - adenosis
        - telangiectasias
    - hypo- or hyperpigmentation of skin
- prognosis
    - mortality 16-55%
        - also depends upon quality of care and rapidity of diagnosis and treatment
    - mortality according to SCORTEN score:
        - 0-1: 3.2%
        - 2: 12.1%
        - 3: 35.3%
        - 4: 58.3%
        - ≥5: 90%

DC11 urticaria

https://dermnetnz.org/topics/urticaria-images/

- background
    - pruritic, elevated skin lesions surrounded by an erythematous base
    - due to transient extravasation of plasma into the dermis
    - 25% of people have it at some stage
    - more common in women

    - deeper subcutaneous extension is rarer and termed angioedema
        - involves face, hands and feet and sometimes other areas (trunk, genitalia, mucous membranes)
    - acute if present for less than 6 weeks, chronic if more
- aetiology
    - due to activation of mast cells in the skin resulting in the release of histamine and other mediators
        - causes capillary leakage and swelling of the skin
        - vasodilation causes the erythema
    - there is thought to be an autoimmune element to chronic urticaria
- acute urticaria
    - cause only identified in about half of cases
    - possible triggers include:
        - allergies
            - foods
            - bites
            - stings
            - medication
        - viral infections
        - skin contact with chemicals, latex, nettles etc.
        - physical stimuli
            - firm rubbing (dermatographism)
            - pressure
            - cold
            - heat
- chronic urticaria
    - chronic spontaneous urticaria
        - triggers include medication, stress, infection
        - previously known as idiopathic urticaria
    - autoimmune urticaria
        - may account for half of cases in adults and older children
        - may have an association with other autoimmune conditions
    - inducible urticaria
        - triggers include:
            - contact with hot or cold water (aquagenic)
            - exercise or emotion (cholinergic)
            - exposure to cold or heat
            - firm rubbing, minor trauma (dermatographism)
            - pressure (delayed pressure)
            - vibration
            - sun exposure (solar)
            - infections (viral, bacterial, parasitic)
            - urticaria prior to menstruation (autoimmune progesterone dermatitis)
- differential
    - erythema multiforme
    - dermatitis herpetiformis

    - pemphigoid
    - erysipelas
    - eczema
    - urticaria pigmentosa
    - chronic pruritus
    - polymorphic eruption of pregnancy
    - urticarial vasculitis
        - inflammation of small blood vessels rather than skin involvement
- investigation
    - usually a clinical diagnosis
    - in chronic or recurring cases, investigations will be guided by history and may include:
        - FBC
        - ESR/CRP
        - physical challenge
            - cold provocation (ice cube)
            - heat provocation (warm water)
            - pressure testing
            - UV light testing
            - exercise or hot bath provocation for cholinergic urticaria
        - elicit dermatographism
        - patch/prick testing for contact urticarias
        - IgE tests for specific allergens
        - thyroid autoantibodies if autoimmune mechanism suspected
        - exclusion of suspected medication or food
        - tests for infectious diseases
        - skin biopsy for urticarial vasculitis
- management
    - identify and treat the cause where possible
    - minimise nonspecific aggravating factors
        - overheating
        - stress
        - alcohol
        - caffeine
        - medication likely to cause urticaria (e.g. NSAIDs, ACEi)
    - topical antipruritic agents
        - e.g. calamine lotion or topical menthol 1% in aqueous cream
    - non-sedating antihistamines
        - usually cetirizine, loratadine or fexofenadine
        - continue for 3-6 months after symptom control achieved
        - doses of up to 4 times the standard dose can be used or another antihistamine added
            - an additional sedating antihistamine may be added if itching interrupts sleep (e.g. chlorphenamine)
        - avoid antihistamines where possible in pregnancy (no good safety data); if required, chlorphenamine is first choice
    - steroids

        - may be used as a short course for severe symptoms (e.g. 40mg PO for 7 days)
    - secondary care options for refractory chronic urticaria:
        - antileukotrienes (e.g. montelukast) combined with an antihistamine
            - little evidence they are effective as a monotherapy
        - omalizumab (anti-IgE antibody)
            - effective in 80%
            - requires monthly injections
            - relapse common when stopped
        - ciclosporin for immunosuppressive effect
- referral
    - required if symptoms not well controlled or antihistamines required continuously for more than 6 weeks to achieve control
    - if painful and persistent will require biopsy to rule out vasculitic urticaria
    - safety netting from ED regarding angioedema and anaphylaxis and advice to follow up with GP for referral for the above reasons
- prognosis
    - variable
    - most idiopathic urticaria resolves over 6 months
        - some persist for years
        - some remit and relapse
    - 50% of cases of chronic urticaria have resolved in 3-5 years
    - at least 20% of chronic urticaria requiring secondary care referral are still symptomatic 10 years after first presentation
        - factors associated with long duration include:
            - severe symptoms
            - associated angioedema
            - positive antithyroid antibodies
- complications
    - insomnia
    - depression
    - poorer quality of life
    - anaphylaxis may occur in association with acute urticaria

EAR, NOSE AND THROAT

EP1 ENT foreign bodies

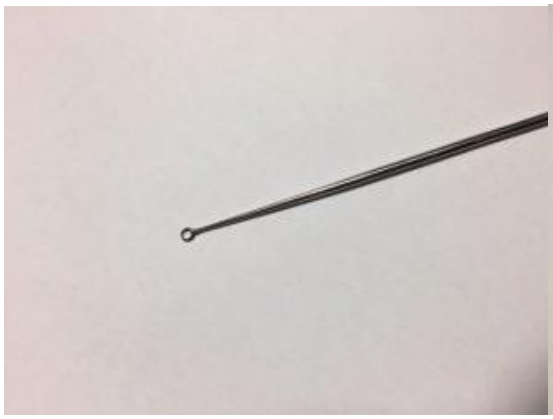
- ear
    - background
        - extraction can be difficult
        - attempts can accidentally cause impaction where the auditory canal narrows
        - 75% of patients with ear foreign bodies are younger than 8
            - usually food or inorganic matter
        - adults most commonly present with live insects in the ear
    - presentation

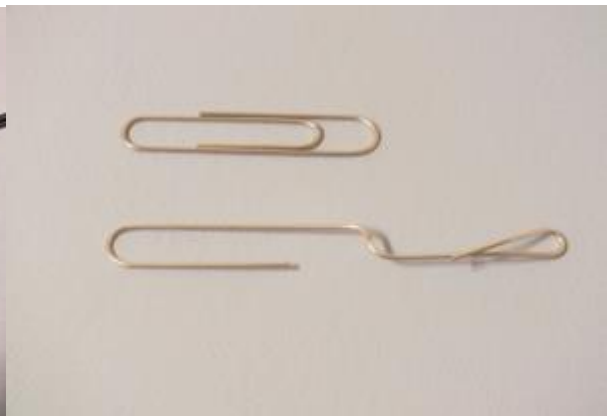
        - children do not always know or are able to tell that a foreign body is present
        - symptoms include:
            - ear pain
            - fullness
            - impaired hearing
        - they may present later with associated otitis media and purulent discharge
        - uncommon but suspicious signs and symptoms:
            - tinnitus
            - vertigo
            - significant hearing loss
            - bleeding
    - management
        - comfortable and secure positioning of patient
        - check whether we should be removing it or referring straight to ENT
            - ENT should help with:
                - button batteries
                    - consider single attempt only – can cause rapid necrosis, do not delay definitive treatment
                - sharp objects
                - tightly wedged foreign bodies not removed after a few attempts
        - check for tympanic membrane perforation
            - may not be visible if foreign body is in the way
            - if present, proceed with removal but do not put any liquids in the ear
                - consider ENT referral as there is a risk of pushing the foreign body through the tympanic membrane into the inner ear
        - consider anaesthetising the ear
            - lignocaine can be dripped in but success rate is low after topical anaesthesia
            - nerve blocks can be considered
                - however if the patient cannot stay still for a retrieval they are unlikely to tolerate a nerve block
                - referral for sedation or general anaesthesia may be required
        - when removal is successful, double check that everything has been removed
            - make sure the other ear has also been checked
        - discharge with safety net advice
        - consider prophylactic antibiotic drops if there has been skin abrasion
        - if the foreign body cannot be removed, ENT review the next day is appropriate unless:
            - severe pain
            - suspected tympanic rupture
            - button battery
    - potential removal methods
        - forceps
            - if the foreign body is ‘graspable’, crocodile forceps could be used
            - best for organic matter

- may be best to hide the forceps from the patient as they can look a bit threatening
- irrigation
  - especially useful for live insects
    - ideally check for tympanic membrane perforation first
      - bear in mind insect may try to fly towards otoscope light if alive which can be uncomfortable for the patient
    - insect must be killed with alcohol, 2% lidocaine or mineral oil
    - suction is usually more effective than grasping for removal to avoid shedding
  - warm water must be used
    - cold water will cause vertigo and vomiting
  - a cannula tube connected to a syringe for slow flushing and good directionality may be helpful
    - avoid directing water towards the tympanic membrane as it will cause perforation
    - it should be aimed at the side of the ear canal – it will swirl around the canal and flush out the foreign body more effectively
  - irrigation should not be used for button batteries or for organic matter, which may swell and become wedged
- modified suction
  - most EDs do not have microsuction
  - cutting a 12F suction catheter short and applying gentle suctioning may be an alternative
    - using the soft tubing cut from a butterfly needle may also work
    - Yankauer catheters are too big for the ear
  - pressure should be set at around 100-140mmHg
    - the patient must be warned that it will be noisy
- glue
  - should not be attempted unless experienced in the technique
  - wound glue on the end of a syringe or Q-tip can adhere to the foreign body and pull it out
  - necessary to be convinced that the foreign body will come out and not just stick further to the ear canal
  - putting an ear speculum on the foreign body can help to guide the glue towards it and protect the rest of the ear canal
  - patient must be compliant
- magnets
  - a small magnet may theoretically remove a magnetic foreign body
  - unlikely to be available in the ED and risk of dropping magnet into ear

- noses
  - background

        - common
        - may theoretically be behind a turbinate, so difficult to see and remove
        - if there is a high clinical suspicion but nothing visible, refer to ENT for nasendoscopy
        - most nasal foreign bodies are in the anterior or middle third of the nose
    - presentation
        - patients may not always mention a nasal foreign body
        - high index of suspicion for unilateral purulent discharge, unilateral sinusitis or recurrent unilateral epistaxis
        - button batteries must be removed as soon as possible and can cause significant injury within hours to days
    - removal methods
        - mother's kiss
            - works in 60% of cases
            - occlude other nostril and ask parent to blow into child's mouth
            - can be done with bag valve mask if needed but ensure pressure not too high
            - older children may be encouraged to blow their own nose
            - Sellick's manoeuvre (cricoid pressure) may be considered to avoid air passage into the oesophagus – this is probably not needed
        - suction
            - gentle suction may help, started slowly and from the inferior part of the nostril
        - curved probe





https://www.rcemlearning.co.uk/foamed/stuck-in-the-ear/

            - jobson horne probe if possible
            - if none available, a loop can be made at the end of a paperclip to use as a scoop
        - foley catheter
            - small catheter size 6-8
            - check balloon is functional
            - insert catheter with balloon deflated past foreign body then inflate balloon with 1-3ml of air and pull gently, avoiding trauma to nose
                - foreign body comes out with catheter

- refer to ENT if unable to remove, as there is a risk of aspiration in sleep if waiting until next day

EP2 ENT injuries

- traumatic perforation of tympanic membrane
  - assessment
    - causes
      - blunt force trauma to external ear canal (e.g. blow to side of head)
      - penetrating trauma (e.g. cotton buds, iatrogenic)
      - barotrauma (e.g. explosions, scuba diving)
    - presentation
      - otalgia
      - hearing loss
      - aural fullness
      - tinnitus
      - serosanguinous discharge
    - examination
      - otoscopy and documentation of perforation (clock face)
      - Rinné and Weber's tests
      - facial and other cranial nerves
  - red flags
    - significant generalised head or polytrauma
    - Battle's sign
    - facial nerve palsy
  - management
    - saline or sodium bicarbonate drops can be used if there is a lot of clotted blood
      - large amount of blood unlikely with isolated tympanic membrane perforation and suggests more extensive injury
    - topical antibiotic drops only if contamination, not for dry uncontaminated perforations
    - advise to keep ear clean and dry to reduce risk of secondary infection
    - advise to place soft ball of cotton wool rolled in petroleum jelly against the canal when showering with soap/shampoo
    - advise GP review if developing discharge/pain
  - outcome
    - isolated, uncomplicated dry perforations usually heal spontaneously with no need for specialist follow-up
    - majority heal within 8 weeks
    - if there is recurrent discharge or noticeably worse hearing they should ask for ENT clinic referral
      - may require surgical repair (myringoplasty)
    - more complex patients need ENT review and follow up
- nasal trauma
  - assessment
    - considerations
      - any serious head injury, other facial fractures, need for CT head

- cleaning and closure of any wounds
- investigation of the cause of the trauma (e.g. cause of collapse)
- whether there is a septal haematoma
    - collection of blood under the lining of the nasal cartilage
    - red/purple fluctuant swelling on both sides of the nasal septum
        - can be confused with a deviated nasal septum – however this would have a concavity on the other side
    - palpation with a Jobson Horne probe for fluctuance
    - haematoma can be confirmed by aspiration of blood/clot with green needle
    - requires prompt ENT review for incision and drainage
    - if left untreated can lead to septal abscess, ascending cavernous sinus infection, necrosis of the septal cartilage, saddle deformity, septal perforation
- whether epistaxis has stopped
- whether there is clear watery discharge after epistaxis stops
    - may present several days later
    - usually unilateral if CSF leak
    - send a few ml to lab for beta-2 transferrin/tau protein
    - usually managed conservatively if no meningism/neurological signs
    - unlikely in isolated nasal trauma with no LOC
    - rarely require endoscopic repair

- nasal fracture
    - x-ray is not indicated
    - if isolated, can be managed as an outpatient (nose is too swollen to assess initially)
    - patients should be seen in the ENT clinic after 7-10 days for consideration of manipulation and further treatment
        - considered if there is a new lateral deviation of the nasal dorsum
        - manipulation should ideally be within 14 days and certainly within 21 days
- red flags
    - associated base of skull fracture/other facial injuries
    - septal haematoma
    - severe traumatic epistaxis requiring packing (90% stop with conservative methods)

- blunt trauma to the neck
    - assessment
        - consider:
            - dysphagia
            - surgical emphysema
            - continuously blood stained saliva
            - hoarseness/loss of voice
            - difficulty in breathing

- noisy breathing
- haemoptysis
- deviated trachea or larynx
- burns:
    - singed eyebrows, facial burns, soot near mouth and nose

- management
    - sedation and intubation if indicated
    - may need flexible endoscopy by ENT
    - CT neck +/- angiography if stable enough
    - admission for observation
    - medical management includes:
        - IV steroids
        - humidification
        - antibiotics if haematoma or surgical emphysema
        - anti-reflux medication
        - voice rest
        - SALT input
        - water soluble contrast swallow test
    - surgical management
        - depends on injury and stability of patient
        - Schaefer classification of airway trauma:
            - group 1
                - minor endolaryngeal haematomas or lacerations without detectable fractures
                - usually conservative management with close airway observation
            - group 2
                - more severe oedema, haematoma, minor mucosal disruption without exposed cartilage, nondisplaced fractures
                - often managed conservatively
                - requires panendoscopy to assess for injuries not seen on nasal endoscopy
            - group 3
                - massive oedema, large mucosal lacerations, exposed cartilage, displaced fractures, vocal cord immobility
                - likely to require tracheostomy for definitive airway prior to surgical correction of injuries
            - group 4
                - same as group 3 but more severe, with disruption of anterior larynx, unstable fractures, two or more fracture lines or severe mucosal injuries
                - requires tracheostomy as well as surgical fixation and stenting
            - group 5

- complete laryngotracheal separation
- high mortality as altered anatomy may make tracheostomy difficult

    - red flags
        - neck trauma with noisy breathing
        - neck trauma associated with laryngeal voice change – hoarse, croaky, husky or no voice
        - expanding swellings in the neck that could be indicative of haematoma
        - any history or signs of head and neck burns – singed eyebrows, mucosal burns, soot in nostrils, swollen lips
- penetrating neck trauma
    - assessment
        - history
            - details from patient/collateral
            - odynophagia, otalgia, voice change
        - initial management
            - c-spine alongside airway if mechanism requires it
            - consider RSI/front of neck access
            - active bleeding
                - firm pressure over the bleeding site
                - avoid compromising the airway
                - avoid using too many layers of swabs – it will obscure the wound and the pressure is applied more diffusely
                - consider use of adjuncts:
                    - Foley catheter inserted into the wound for tamponade
                    - haemostatic granule dressing (e.g. Celox)
            - bubbling or sucking neck wounds
                - place occlusive dressing with diffuse pressure
                - place head down on left side if necessary
            - other initial management steps:
                - nil by mouth
                - humidified oxygen or frequent saline nebs
                - flexible nasendoscopy to assess for blood, clots, mucosal disruption
                - antibiotics if grossly contaminated wound or significant risk of visceral injury
            - CT angiography of the neck if possible
            - ideally remove foreign objects in theatre as risk of severe bleeding
    - classification of penetrating neck injuries
        - velocity
            - high velocity (e.g. firearm projectiles)
            - low velocity (e.g. stab injury)
        - anatomical zone
            - zone 1 – clavicles to level of cricoid cartilage
                - contents are generally less frequently injured but carry highest mortality risk

        - contents are: subclavian artery and vein, apex of lung, brachial plexus, oesophagus, trachea etc.
    - zone 2 – level of cricoid cartilage to angle of mandible
        - generally the most injured area
        - beware of trauma to larynx, pharynx, carotid, vertebral arteries, jugular veins, cranial nerves X/XI/XII
    - zone 3 – angle of mandible to skull base
        - injuries are rarer
        - contents include: the great vessels, pharynx, spinal cord, cranial nerves XI/XII
- further management
    - smaller superficial wounds may be washed and closed in the ED
    - significant/deeper wounds should be explored in theatre
        - e.g. rigid endoscopy, washout, repair, drains, closure
    - observation before discharge is often required
    - consideration of:
        - antibiotics
        - anti-reflux medication
        - interval flexible nasendoscopy
        - voice rest
        - SALT input
        - water soluble contrast fluoroscopic swallow
- red flags
    - neck trauma with noisy breathing or laryngeal voice change – hoarse, croaky or no voice
    - expanding neck swellings that could be haematomas
    - neck wound with profuse active bleeding, massive subcutaneous emphysema, air bubbling from the wound or frank haemoptysis
    - neck wound in a patient with cardiovascular instability

EP3 epistaxis

- background
    - common issue, up to 1 in 200 presentations to the ED in the US
    - many attendances could be avoided with knowledge of first aid
        - this is compounded by a lack of first aid training, equipment and epistaxis teaching for ED staff
    - a co-operative and well-defined ED strategy can result in 85% of patients with epistaxis being managed by ED clinicians
- definition
    - haemorrhage from the nostril, nasal cavity or nasopharynx
    - anterior epistaxis makes up 95% of cases
    - can be divided into traumatic or non-traumatic
        - traumatic usually implies significant external injury to the nose
        - a high percentage of non-traumatic epistaxis is caused by local minor trauma to the nasal mucosa such as picking or rubbing
- pathophysiology

- the anterior nasal septum is the location of a number of arterial anastomoses between vessels arising from branches of the internal and external carotid arteries
    - the series of anastomoses are formed into a triangle shape and known as Kiesselbach's plexus
    - the area of the nasal septum involved is known as Little's area
- the vessels in this area play a large role in thermal regulation, humidification of inhaled air and control of the lumen of the nasal passages
- bleeding not arising from Kiesselbach's plexus (posterior epistaxis) may originate from any part of the remainder of the nasal cavity or nasopharynx
- mechanisms by which most epistaxis occurs is poorly understood
    - presentations are more common in the winter months, probably related to upper respiratory tract infections and lower humidity
    - there are well-defined peaks at extremes of age which may represent different aetiology
    - it is hypothesised that crusting of the mucosa and subsequent

- epistaxis in children may be related to staphylococcal colonisation of the anterior nasal cavity
    - in adults there is an association with recent (in past 24 hours) heavy drinking or regular high alcohol intake – probably due to the effect of alcohol on bleeding time
        - coagulopathies or medications affecting haemostasis also increase risk
        - other factors increasing epistaxis risk include:
            - surgery
            - local malignancy and aneurysms
            - nasal septal deviation
            - use of drugs acting on the nasal mucosa, including cocaine
        - the association of epistaxis with hypertension is more unclear – a large population study has not found a link between the two
- resuscitation and first aid measures
    - assessment of haemodynamic status
        - important even if bleeding appears to have stopped
        - if actively bleeding, may require resuscitative measures alongside first aid
        - treat as a circulatory emergency if indicated
            - especially in elderly, patients on anticoagulation, patients with bleeding disorders
            - insert at least a green cannula
            - send FBC, U&E, coagulation and G&S
            - try to locate the patient in majors or an area where they can be seen – dislodgement of a clot can lead to catastrophic bleed
    - first aid measures
        - lean patient forwards in upright position to avoid passage of blood into the nasopharynx
            - encourage the patient to spit out any blood passing into the throat and not swallow it
        - ask the patient to firmly pinch the soft part of the nose, compressing the nostrils, for at least 10 minutes

                - if patient is unable to comply, this can be done by a family member or member of staff or using an external device such as a swimmer's nose clip
            - ice
                - sucking on an ice cube reduces nasal blood flow
                - applying an ice pack directly to the nose may help
                - application of ice to the neck or forehead has not been shown to reduce nasal blood flow
- history
    - side, duration and approximation of amount of bleeding
    - history of trauma
    - previous episodes and treatment
    - recent upper respiratory tract infection or rhinitis
    - presence of bleeding or bruising elsewhere on the body
    - other significant co-morbidities, particularly those which may affect coagulation such as liver disease
    - medication, particularly drugs which affect coagulation
    - use of drugs of abuse, particularly cocaine
    - alcohol history
    - possibility of foreign body in nose in children
- examination
    - general examination of cardiovascular and respiratory status
    - evidence of signs suggesting pre-existing coagulation disorder
        - e.g. petechiae, purpura, hepatosplenomegaly, lymphadenopathy
    - examine oropharynx with help of tongue depressor if the bleeding stops or history is suggestive of posterior bleed
    - examine nostrils for blood clots with bowl and suction apparatus ready as dislodging clots can lead to rebleed
- risk stratification
    - no validated risk stratification exists
    - if the patient is normally fit and well, with no significant comorbidity or evidence of coagulopathy and no haemodynamic instability, they do not require blood tests
    - routine coagulation tests are not required unless there is a personal or family history of a coagulation disorder
- management
    - equipment and personal protection
        - there is a high risk of blood contamination due to bleeding into the airway and likelihood of droplet spread
        - clinicians should be equipped with a minimum of gloves, mask and visor
        - covering the patient's mouth with a facemask also helps to contain any coughed or expectorated blood
        - essential items for managing epistaxis:
            - strong light source
            - suction apparatus
            - combined anaesthetic and vasoconstrictor agent (e.g. lidocaine with phenylephrine)
            - nasal speculum

        - method of cautery (silver nitrate stick or equipment for electrocautery)
- nasal cautery
    - obtain verbal consent
    - clear the anterior nose with gentle suction
        - a cut down flexible suction catheter can be less traumatic than a rigid catheter
    - identify the site of bleeding and apply local anaesthetic/vasoconstrictor by spray or on cotton wool pledget
        - after it takes effect there should be a clearer view of the anterior nasal cavity
    - if a visible vessel or localised area of bleeding is seen, it should be cauterised
        - either by direct application for no more than 30 seconds in one spot
        - or, if bleeding is too brisk, by cauterising the four quadrants immediately around it, doughnutting the bleeding site
    - silver nitrate and electrocautery have been found to be equally effective
    - only one side of the septum must be cauterised
        - if both sides are cauterised at the same time there is a risk of septal perforation due to decreased vascular supply from the perichondrium
    - excess silver nitrate can be removed by application of a saline soaked pledget to the area, which neutralises the silver nitrate, preventing staining and unwanted areas of burning
    - the patient should be observed for 15 minutes after application prior to discharge
- topical treatment
    - topical antiseptic cream (e.g. Naseptin) is as effective as silver nitrate cautery in preventing further nosebleeds in children with recurrent epistaxis
    - most of the time, nosebleeds in children will stop with simple first aid measures and reveal either nasal crusting or a visible vessel
    - children with recurrent nosebleeds and nasal crusting should be treated with topical nasal antiseptic cream twice daily for 4 weeks
    - in the presence of a visible vessel, silver nitrate cautery is recommended
    - in adult patients, particularly those on antiplatelets (e.g. aspirin), treatment with a topical application of a TXA soaked cotton pledget (500mg TXA in 5ml) is a painless, rapid and effective way of achieving haemostasis in anterior epistaxis that does not stop with direct pressure
        - can be tried prior to the placement of an anterior nasal pack
- nasal packing
    - if first aid measures and attempts at cautery are unsuccessful or there is bilateral bleeding, the nose should be packed
    - there are two main types of nasal tampon in use, compressed sponge (e.g. Merocel, Rhino Rocket) and inflatable balloons (e.g. Rapid Rhino)
        - Rapid Rhinos are the least painful and the easiest to remove
    - once an anterior pack has been placed, the patient should be observed for at least 30 minutes to check no further leakage occurs form the nose or into the pharynx posteriorly

        - there are two sixes of Rapid Rhino pack (5.5.cm and 7.5cm)
            - they should be immersed in saline or distilled water before insertion
            - air should be injected to the amount tolerated or required for haemostasis and no more as this will cause discomfort and possibly pack trauma
        - all anterior nasal packs should be placed as horizontally as possible
        - for Merocel packs, antiseptic cream should ideally be applied before insertion and once inserted a few ml of saline or distilled water should be instilled for the pack to expand
        - the threads of Merocel or tube end of Rapid Rhino should be secured to the face with tape
            - the oropharynx should then be re-examined for posterior bleeding
        - failure of an anterior pack to stop epistaxis is usually due to a posterior bleed
            - management options for this include:
                - endoscopy with cauterisation
                - ligation of the sphenopalatine artery
                - posterior packing
            - if there is significant delay to specialist input and the patient's haemodynamic status is deteriorating, Foley catheters can be used as a temporary solution
                - size 12 or 14 catheters should be advanced one at a time through the nostril, along the floor of the nose into the nasopharynx until seen in the pharynx
                - each balloon should be inflated with 5-10ml water and gentle traction applied
                - it is a technique of last resort when specialist help is unavailable
- admission after packing
    - traditionally all patients were admitted after packing due to fears of potential airway compromise with ribbon gauze insertion
    - certain groups of patients with nasal tampons can be discharged with early review in the ENT clinic
    - patients with anterior packing requiring admission include:
        - traumatic cause for the epistaxis
        - haemodynamic compromise or shock
        - previous nasal packing within the past 7 days
        - patient taking anticoagulation
        - measured haemoglobin <100
        - uncontrolled hypertension
        - significant co-morbid illness
        - adverse social circumstances (e.g. patient lives more than 20 minutes from the hospital or has no access to telephone or transport)
        - patient's personal preference
    - no follow up is required for patients in whom the epistaxis has stopped spontaneously or by first aid measures or cautery alone

        - patients should be provided with advice to prevent recurrence and first aid measures
            - they should be advised to avoid:
                - blowing the nose for one week
                - sneezing through the nose – they should keep the mouth open
                - hot and spicy drinks and food, including alcohol for two days
                - heavy lifting, straining or bending over
                - vigorous activities for one week
                - picking the nose
        - for patients with an anterior nasal pack, it should be left in place for 24-48 hours, then they should be seen by ENT for removal and further assessment
            - there is no evidence for routine antibiotic cover

EP4 hearing loss

- background
    - sudden onset hearing loss is a subjective symptom in one or both ears, perceived by the patient
    - a pure tone audiogram must be performed to objectively determine if there is hearing loss
    - typically occurs within a few days
    - alarming symptom, and sometimes a medical emergency
    - may be conductive or sensorineural
    - all patients require urgent assessment
    - definition is hearing loss of 30dB or more over at least three contiguous frequencies over a period of 72 hours or less
- aetiology
    - outer ear (conductive hearing loss)
        - foreign body
        - wax
        - otitis externa
        - other ear canal pathology (e.g exostoses)
        - trauma (syringing)
    - middle ear (conductive hearing loss)
        - otitis media with effusion
        - haemotympanum
        - ossicular chain discontinuity
        - trauma
        - barotrauma
        - iatrogenic (post-operative)
        - tympanic membrane perforation
        - cholesteatoma
    - inner ear (sensorineural hearing loss)
        - idiopathic
        - infective
            - viral/bacterial (e.g. HIV, CMV, HSV, mumps, rubella, syphilis)
        - noise induced

        - trauma (temporal bone fracture)
        - ototoxic drugs
        - autoimmune
            - SLE, granulomatosis with polyangiitis, relapsing polychondritis, ulcerative colitis)
        - tumour
            - vestibular schwannoma, leukaemia, myeloma
        - vascular
            - cerebrovascular disease, sickle cell disease
        - perilymphatic fistula
        - barotrauma
        - neurological
            - multiple sclerosis, CVA, migraine
        - other
            - diabetes, sarcoidosis
        - non-organic hearing loss
- red flags associated with sudden onset hearing loss
    - concurrent head trauma
    - neurological signs or symptoms
    - unilateral middle ear effusion (post-nasal space must be examined)
- history
    - which ear is affected
        - bilateral sudden hearing loss is rare, but can be caused by autoimmune disease, syphilis, trauma, neoplasia and vascular causes
    - clarify whether it was truly sudden loss or more gradual
    - any previous problems with that ear
    - any activity being undertaken at the time of the hearing loss
        - water may precipitate wax impaction
    - tinnitus and dizziness are non-specific and may not help to differentiate between conductive and sensorineural loss
    - coryzal symptoms, fever, discharge, otalgia or recurrent infections with discharge point to a cause
    - past medical history
        - including autoimmune disease, diabetes, sarcoidosis, vascular disease
    - excess noise exposure or acoustic trauma
    - previous otological surgery
    - medication history
        - including history of ototoxic drugs
            - e.g. aminoglycosides, furosemide, NSAIDs, chemotherapy agents, quinine, high dose salicylates
    - relevant family history such as otosclerosis
- examination
    - lymphadenopathy
        - may indicate malignancy or middle ear infection affecting the facial nerve
    - cranial nerve abnormalities
        - may suggest intracranial lesions such as acoustic neuroma or malignancy, or multiple sclerosis

    - discharge at the external meatus or pain on moving the pinna
        - suggests otitis externa
    - mastoid tenderness or fluctuance
        - suggests mastoiditis
    - otoscopy
        - using a systematic approach, starting from the external meatus medially
        - look for:
            - foreign bodies
            - wax
            - discharge
            - masses
            - retraction of the eardrum
            - evidence of middle ear effusion
            - tympanic membrane perforation
    - tuning fork tests
        - to differentiate type of hearing loss
        - a 512 Hz tuning fork is most reliable
        - Rinné test is positive when air conduction is better than bone conduction in that ear (normal test) and negative when bone conduction is louder
            - sensitivity is low
    - free field testing
        - examiner tests hearing with whispered, normal and loud voice (indicating higher sound thresholds) while standing 60cm behind the seated patient
        - if responses are poor, it can be repeated at 15cm
        - does not differentiate between types of hearing loss
        - reported sensitivity is 90-100% and specificity 70-87% in adults
- investigations
    - likely to be led by ENT
        - pure tone audiogram
            - determines whether there is hearing loss and the degree and type
        - tympanology
            - assesses tympanic membrane mobility and inner ear function
        - flexible nasendoscopy
            - visualises the postnasal space for posterior masses
    - blood tests may include:
        - FBC
        - U&E
        - ESR
        - glucose
        - cholesterol
        - viral titres (HIV, CMV, HSV, mumps, rubella)
        - VDRL or equivalent for syphilis
        - Lyme titres
        - TFTs
        - ACE
        - ANCA, ANA, RF, anti-CPP, anti-phospholipid antibodies
    - imaging

        - MRI with gadolinium contrast of the internal acoustic meatus and brain
            - for unilateral or asymmetrical sensorineural hearing loss to exclude vestibular schwannoma
            - may also identify other causes such as multiple sclerosis or small vessel ischaemic changes
        - CT of the temporal bones
            - may be used in patients with contraindications to MRI
            - can exclude large acoustic neuromas and evaluate middle ear/ossicular chain in conductive hearing loss
        - CXR
            - if there is a suspicion of sarcoidosis with mediastinal involvement
- management
    - conductive hearing loss
        - can often be managed by GP
            - e.g. wax, otitis externa
        - referral required for evaluation of the postnasal space if there is unilateral effusion
        - tympanic membrane perforations usually heal spontaneously within a few days to weeks
            - topical antibiotics not required unless there is an associated infection
            - referral to ENT for consideration of repair if the perforation does not heal
    - idiopathic sensorineural hearing loss
        - diagnosis of exclusion
        - indicators of better prognosis include:
            - low frequency hearing loss
            - less severe hearing loss at presentation
            - early treatment
        - mainstay of treatment is early oral steroids (1mg/kg/day up to 60mg) unless contraindicated
            - individual risk benefit analysis usually still favours steroids in diabetic patients, with careful monitoring and management of blood sugars
            - treatment is usually 7-14 days, tapering not required for shorter courses
            - they have not shown a conclusive benefit
            - if steroid treatment fails, early referral should be made
            - if steroid treatment is to be started, a risk benefit discussion should be had with the patient and a discussion with ENT is warranted
            - the patient will require referral for PTA and ENT review
    - other causes
        - require ENT referral

EP5 painful ear

- causes of otalgia
    - external ear causes

        - otitis externa
        - foreign body
            - including live insects
        - trauma
        - impacted cerumen
        - bullous myringitis
        - furuncle
        - herpes zoster
        - neoplasm
        - otomycosis
        - perichondritis of pinna
        - Sjögren's syndrome
    - middle ear causes
        - otitis media
        - effusion associated with otitis media
        - acute mastoiditis
        - barotrauma
        - acute obstruction of the Eustachian tube
        - neoplasm
        - trauma
    - referred pain
        - nasopharynx
            - adenoidectomy
            - infection
            - neoplasm
        - cranial nerve referred pain
            - trigeminal neuralgia (V)
            - Ramsay Hunt syndrome (VII)
            - glossopharyngeal - -tonsillitis
        - salivary glands
            - calculi
            - infection
        - teeth and jaw
            - impaction of molars
            - malocclusion
            - temporomandibular joint arthritis
        - base of skull
            - elongated styloid process
        - petrous aneurysms
        - oesophagus
            - foreign body
            - reflux
            - neoplasm
        - inflammation or neoplasm of oropharynx, tongue or larynx
        - temporal arteritis
        - thyroiditis
- approach to patient with otalgia

    - history
        - onset
        - precipitating factors
            - e.g. noise
        - duration
        - discharge
        - fever
        - swallowing disorder
        - dental history
    - examination
        - e.g. otitis media, cerumen
        - if otoscopy unremarkable, consider referred causes of pain and examine cranial nerves (especially V, IX, X)
        - examination of nose, sinuses, oropharynx, nasopharynx, TMJ, parotid glands, larynx, trachea
        - check temperature
    - investigations
        - depend on suspicion from history and examination
        - consider:
            - FBC
            - TFTs
            - ESR
            - CXR
            - audiography
    - always consider neoplastic causes in children and adults with persistent otalgia
        - other red flags include weight loss, voice change, lymphadenopathy, dysphagia
- management
    - analgesia
    - treat underlying cause
    - safety netting for consideration of GP review in a few days if no cause found
- prognosis
    - around 50% have spontaneous resolution with no underlying cause detectable

EP6 sore throat

- background
    - common
    - usually viral
    - most common bacterial agent is Group A beta-haemolytic streptococcus
        - accounts for 20-40% of sore throats in children, 10% in adults
- history
    - duration and severity of symptoms
    - any self medication/OTC treatment
    - comorbidities, previous risk factors, relevant past history
    - feeling systemically unwell
    - dysphagia
    - rash

    - stridor
    - typical symptoms such as fever, headache, malaise, rhinitis, cough, hoarseness
- examination
    - features of epiglottitis such as drooling, leaning forward, high temperature
    - examination of throat with tongue depressor
        - redness of pharynx and tonsils
        - enlargement of tonsils
        - presence of exudate
    - enlarged tender cervical lymph nodes
    - scarlet fever-like rash (red punctate skin eruption prominent in the skin creases), flushed face, circumoral pallor and strawberry tongue suggest possibility of streptococcal infection
    - recurrent sore throat with fever and lymphadenopathy may suggest glandular fever
- Centor criteria
    - developed to help predict bacterial infection clinically
    - score out of 4
        - score of 3 or 4 suggests possibility of GABS is 40-60% and antibiotic therapy may be beneficial
        - patients without 3 or 4 signs have an 80% chance of a viral infection
    - components:
        - tonsillar exudate
        - tender anterior cervical lymph nodes
        - absence of cough
        - history of fever
- investigations
    - not normally required
    - FBC and glandular fever test if glandular fever suspected
- management
    - reassurance
    - paracetamol/ibuprofen
    - adequate fluid intake
    - consideration of throat lozenges, hard boiled sweets, ice, ice lollies for symptomatic relief
    - safety netting for urgent review if:
        - breathing difficulty or stridor
        - drooling
        - muffled voice
        - severe pain
        - dysphagia
        - unable to swallow adequate fluids
        - systemically unwell
    - antibiotics should be considered for people who:
        - are systemically unwell
        - have signs of serious illness and/or complications such as peritonsillar abscess or cellulitis
        - are immunosuppressed
        - have valvular heart disease
        - have a significant comorbidity

    - antibiotics shorten duration of pain symptoms by about 1 day
        - they can reduce the chance of rheumatic fever by more than two thirds in communities where it is common, however in the UK the number needed to treat is much higher and there are implications to cost and antibiotic resistance
        - other complications such as otitis media, quinsy and sinusitis are also reduced through antibiotic use
    - when an antibiotic is required, first line is phenoxymethylpenicillin for 10 days, with erythromycin or clarithromycin for 5 days as second line
    - a single dose of 10mg oral dexamethasone for adults and 0.6mg/kg (max 10mg) for children can provide symptomatic relief for pain with few adverse effects
- referral for tonsillectomy criteria
    - adults
        - five or more severe (disabling and causing loss of normal ability to function) episodes per year of sore throat due to tonsillitis (clinically documented, not just reported sore throat)
    - children
        - five or more episodes of acute sore throat per year (documented by parent or clinician) where:
            - symptoms have been a problem for at least a year
            - episodes are severe (disrupt normal behaviour or functioning)
            - advantages and disadvantages have been discussed, including the possibility of spontaneous resolution
            - children have sleep apnoea with daytime drowsiness and failure to thrive
- complications
    - suppurative
        - otitis media
        - sinusitis
        - peritonsillar abscess
        - cervical adenitis
        - mastoiditis
        - scarlet fever
        - streptococcal toxic shock syndrome (rare)
        - Lemièrre's syndrome
            - rare acute septicaemia and jugular vein thrombosis secondary to infection with Fusobacterium spp)
    - nonsuppurative
        - rheumatic fever (rare in developed countries, prevalent in developing countries)
        - post-streptococcal glomerulonephritis
        - guttate psoriasis (may flare up in presence of streptococcal infection)
- prognosis
    - 90% of sore throats are better within a week (with or without antibiotic treatment and whether viral or bacterial)
    - in 40% symptoms have settled within 3 days

EP7 vertigo

- background
    - common problem, affects around 5% of adults/year
    - defined as an illusion of rotatory movement
    - always implies an imbalance in the vestibular system but not where it originates
    - classification of dizziness
        - vertigo
            - an illusion of motion of either the subject or the environment
        - presyncope
            - a feeling of impending faint or loss of consciousness
        - disequilibrium
            - impaired balance and gait in the absence of vertigo, often a sense of impending fall
        - light-headedness
            - non-specific symptoms that cannot be identified as one of the other types
    - vertigo accounts for around 30% of dizzy patients in primary care
        - 43% of presentations to the ED with dizziness were due to a peripheral vestibular disorder
    - vertigo is a symptom not a diagnosis
    - it can be divided into:
        - physiological vs pathological
        - central vs peripheral
- pathophysiology
    - equilibrium relies on sensory inputs from the vestibular apparatus, visual system and proprioceptive stimuli from the neck and the rest of the body
    - the membranous labyrinth is contained within the bony labyrinth lying in the petrous temporal bone
        - connects via the vestibulocochlear nerve to the vestibular nuclei in the brainstem
        - these nuclei interconnect with neurones in the cerebellum, spinal cord and cerebral cortex
    - the membranous labyrinth consists of the three semicircular canals and two chambers, the saccule and utricle
    - flow of endolymph in the canals stimulates cilia attached to a sensory organ located in the ampulla of each canal, the crista amupullaris
    - in the saccule and utricle, movement of calcified calcium carbonate crystals (statoconia or otoliths) stimulates cilia of another sensory organ, the macula
    - movement of the head in any plane modifies neural impulses transmitted via the vestibular nerve, connected to each of the sensory organs, to nuclei in the brainstem
    - vertigo results from an imbalance of either the received signals or information processing in the brainstem
    - nystagmus may indicate the cause of vertigo
        - consists of an initial smooth movement in one direction followed by a rapid movement (saccade) in the opposite direction
        - it is described according to the direction of the fast component

        - can be physiological (e.g. when looking out of the window of a moving train) or pathological
        - a few beats are normal on extreme lateral gaze
- history
    - description of symptoms
        - subjective – I feel like I am moving/spinning
        - objective – it feels like the world is moving/spinning
    - clues for differentiating between peripheral and central vertigo
        - peripheral vertigo
            - onset
                - sudden
            - nausea and vomiting
                - severe
            - effect of head position
                - worsened by position
                - often single critical position
            - associated auditory findings (e.g. aural fullness, tinnitus, hearing loss)
                - may be present
            - associated neurological symptoms (e.g. dysarthria, diplopia, hemiparesis)
                - none
        - central vertigo
            - onset
                - occasionally very sudden but usually gradual
            - nausea and vomiting
                - variable – usually minimal
            - effect of head position
                - little change
                - associated with more than one position
            - associated auditory findings
                - rare
            - associated neurological symptoms
                - usually present
    - other useful factors
        - is it a new event, or is there a history of recurrent episodes
            - short spells of sudden onset vertigo associated with a change in head position are likely to be BPPV
        - past history of vascular disease, hypertension or stroke
            - increases likelihood of central cause
        - recent trauma or infection of the ear
            - increases likelihood of peripheral cause
        - drugs associated with vertigo:
            - ACEi
            - amiodarone
            - aminoglycosides
            - beta blockers

                - cocaine
                - phenytoin
                - salicylates
                - sildenafil
- examination
    - general examination
        - particularly cardiovascular for evidence of vascular disease or atrial fibrillation
    - neurological examination
        - general examination
            - may reveal cranial nerve or peripheral deficits associated with a cerebrovascular event or neoplastic lesion
            - limb ataxia suggests cerebellar disease
    - ears
        - evidence of vesicles (Ramsay Hunt syndrome)
        - tympanic perforation
        - cholesteatoma
    - hearing
        - gross testing with ticking wristwatch/whisper/rubbing fingers together close to each ear
        - if reduction in hearing found, proceed to Weber's and RInné's tests looking for evidence of sensorineural hearing loss
- HiNTS examination
    - used to differentiate between central and peripheral causes
    - only valid in patients who have continuous, ongoing vertigo at the time of assessment
    - components
        - head impulse
            - the patient fixates on the examiner's nose and their head is rotated 20-40 degrees in each direction before being rapidly brought back to neutral
            - normal response is to maintain a continuous direction of gaze
                - this is preserved in posterior stroke
                - in peripheral causes, the vestibular-ocular reflex is disrupted and they lose eye contact and correct with a saccade
        - nystagmus
            - patient is asked to look straight ahead, to the left and to the right whilst direction of nystagmus is observed
            - nystagmus due to a peripheral cause is always horizontal and will always have the fast phase in the same direction
                - often accentuated when the patient looks in the direction of the fast phase
            - any vertical or rotatory nystagmus or change in direction suggests a central cause of vertigo
        - test of skew
            - patient fixates on examiner's nose; the eyes are alternately covered

        - in a central cause, the vertical alignment of the eyes may be different and a vertical corrective movement is seen when the eye is uncovered
- acute vestibular neuritis
    - commonest cause of prolonged peripheral vertigo
    - typically occurs in young and middle aged adults
    - postulated to be caused by viral infection, possibly herpes simplex
    - caused by acute inflammation of the vestibular nerve
    - presentation (typical of peripheral vertigo):
        - acute onset within minutes/hours
        - exacerbated by movements of the head
        - accompanying severe nausea and vomiting
        - no other neurological deficit
        - no disturbance of hearing
    - spontaneous nystagmus is found in two thirds and is peripheral in character:
        - horizontal
        - unidirectional
        - suppressed by visual fixation
        - fast phase away from the affected ear and most evident when looking to the unaffected side
- cerebellar stroke
    - patients are usually older with a pre-existing risk factor such as atrial fibrillation or diabetes
    - onset of symptoms is hyperacute, within a few seconds
    - vertigo is central in character:
        - unaffected by head position
        - little systemic upset
        - generally has co-existing neurological deficits such as ataxia or depressed level of consciousness
    - nystagmus is typically central:
        - horizontal, rotatory or vertical
        - bidirectional
        - not suppressed by visual fixation
- otomastoiditis
    - acute and chronic ear infections may either directly infect or release toxins into the labyrinth causing acute labyrinthitis and peripheral vertigo
    - patient normally complains of fever, ear pain, headache and hearing loss
    - otoscopy reveals evidence of infection
- transient ischaemic attack
    - rarely presents as an isolated central vertigo
    - if it does, temporary features are identical to those of cerebellar stroke
- benign positional paroxysmal vertigo (BPPV)
    - very common
    - caused by dislocation of statoconia into the posterior semicircular canal
    - vertigo is related to changes in head position, e.g. turning over in bed or reaching upwards
    - typically affects women between 60 and 70

    - vertigo is peripheral and short lasting with fatiguing nystagmus in one direction
    - diagnosis is confirmed by the Hallpike manoeuvre
    - it can be treated with the Epley manoeuvre, which can be effectively self administered once taught
- migraine
    - typically presents with episodic headaches accompanied by photophobia, nausea and vomiting
    - vertigo with either central or peripheral features may occur in up to 25% of patients with acute migrainous vertigo
- Ménière's disease
    - commonest cause of acute paroxysmal vertigo with hearing loss
    - caused by an increase in the pressure and volume of endolymph
    - patients normally present with an initial feeling of aural fullness, followed by increased tinnitus, fluctuating hearing loss and peripheral vertigo
    - clinical diagnosis, all investigations are to rule out other diagnoses
- acoustic neuroma/vestibular schwannoma
    - benign tumour of myelin forming cells of the vestibulocochlear nerve
    - gradual progressive hearing loss and tinnitus are common symptoms
    - episodic central vertical may also be a feature
- management
    - key to ED management is distinguishing peripheral from central vertigo
        - all patients with central vertigo require investigation as an inpatient or urgently as an outpatient
        - if peripheral vertigo is diagnosed, most patients can be discharged home with arrangements for follow up with their GP or in an appropriate ENT clinic provided they have an appropriate carer to look after them
            - some patients may benefit from occupational therapy/social services review
            - a few may require admission for IV fluids if unable to tolerate oral fluids even after treatment
    - in all cases of prolonged vertigo, both the sensation of vertigo and the nausea and vomiting are distressing to the patient
        - vestibular suppressants and antiemetics are the mainstay of treatment
            - vestibular suppressants:
                - low dose benzodiazepines
                    - e.g. 2mg diazepam, 0.1mg lorazepam
                - anticholinergics
                    - e.g. hyoscine
                - antihistamines with anticholinergic properties
                    - e.g. cinnarizine, cyclizine
            - antiemetics such as prochlorperazine and metoclopramide are commonly used but may be associated with acute dystonic reactions
            - it is widely thought that recovery from or brain adaptation to shortlasting peripheral vestibular dysfunction such as vestibular neuritis may be hampered by immobilisation and the continued use of vestibular suppressants
        - physical therapies
            - vestibular exercises have been shown to significantly improve symptoms and function for peripheral causes of vertigo (when compared to controls or no intervention)

- patients with anything other than mild, short-lived, self-limiting vertigo because of vestibular dysfunction may benefit from vestibular exercises
    - patients with acute peripheral vertigo can be advised to focus on an object while moving their head side to side then up and down
    - movements should be slow and slight to start with then gradually increased and repeated for several minutes 2-3 times a day

- specific management
    - BPPV
        - canalith repositioning manoeuvres to move canaliths out of the semicircular canals
        - Epley's manoeuvre
            - used to treat BPPV due to posterior canal canaliths (>90% of cases)
            - safe and effective, at least in the short term, and can be done in the ED
            - recurrence rates may be as high as 50% over 5 years – the manoeuvre can be repeated or taught
            - anterior canal BPPV is rare and can be a complication of canalith repositioning manoeuvres
            - Epley manoeuvre:
                - patient is laid flat with their head hanging down over a pillow or end of the trolley (with caution in the elderly or patients with neck problems) with the affected ear downwards
                    - position is held for 30 seconds and results in symptoms and nystagmus (positive Dix-Hallpike)
                - when the nystagmus has stopped, the patient's head is rotated to the other side (affected ear upwards) and held for another 30 seconds
                - the patient is then turned onto the unaffected side with their nose pointing to the floor for another 30 seconds
                - the patient is then helped to a sitting position, keeping their head turned to the unaffected side
                - the process is repeated until nystagmus can no longer be induced
    - vestibular neuritis
        - corticosteroids (methylprednisolone) given acutely may improve longer term vestibular function in patients with presumed viral vestibular neuritis
    - Ménière's disease
        - betahistine or diuretics are often prescribed for dizziness symptoms and are generally well tolerated
            - evidence of efficacy is lacking
        - specialist treatments such as intratympanic gentamicin may be considered in selected cases

EC1 croup

- pathophysiology of stridor
    - children have a compliant chest wall as the ribs are cartilaginous and lie more horizontally than in an adult
    - the accessory muscles are immature and the diaphragm can become easily fatigued
    - an increase in respiratory effort causes tracheal tug, intercostal muscle indrawing and sternal recession, all of which reduce mechanical efficiency
    - children have a small functional residual ling capacity with little respiratory reserve – when there is increased oxygen demand and metabolic demand, decompensation can occur quickly
    - stridor is a harsh vibratory sound produced by turbulent airflow through the respiratory passages
        - normally heard on inspiration but can be heard on expiration or be biphasic
    - inspiratory stridor is normally caused by an obstruction above the glottis due to collapse of the soft tissues
    - expiratory stridor indicates a tracheobronchial obstruction
    - biphasic stridor suggests a fixed subglottic or glottic anomaly, commonly laryngomalacia, less commonly by vocal cord paralysis, subglottic haemangioma or vascular ring
    - the volume of stridor does not correlate with the degree of obstruction
        - a progressive decrease in volume may indicate an increasing obstruction with imminent complete obstruction or resolution of the obstruction
    - underlying pathology is inflammation of the pharynx, larynx, trachea or bronchi
    - Poiseuille's law states that if the radius of the airway is halved, the resistance in airflow increases by 16 times
    - it is the subglottic inflammation and swelling that compromises the airway in croup
- epidemiology
    - around 80% of children presenting with stridor and cough have croup
    - croup is a clinical syndrome of hoarse voice, harsh barking cough and acute inspiratory stridor
    - croup occurs in 2% of children between 6 and 36 months with peak incidence at 12-24 months
    - male to female ration is 3:2
    - more common in spring and autumn but can occur at any time of year
    - viral in 80% of cases
        - usually parainfluenza virus
        - can also be caused by adenovirus, RSV, measles, coxsackie, rhinovirus, echovirus, reovirus and influenza A and B
- presentation
    - typically preceded by a coryzal illness with croup developing over several days
    - symptoms are classically worse at night and typically last 3-5 days but can last up to a week
- Westley croup score
    - interpretation:
        - mild (0-2)
        - moderate (3-5)
        - severe (6-11)

        - impending respiratory failure (12-17)
    - scoring
        - stridor
            - none (0)
            - upon agitation (1)
            - at rest (2)
        - retractions
            - none (0)
            - mild (1)
            - moderate (2)
            - severe (3)
        - air entry
            - normal (0)
            - mild decrease (1)
            - marked decrease (2)
        - sats <92%
            - none (0)
            - upon agitation (4)
            - at rest (5)
        - level of consciousness
            - normal (0)
            - decreased (5)
    - 85% have mild croup
    - around 5% require admission and 1-3% of these require intubation
- management
    - initial measures
        - keep the child comfortable and avoid agitation
        - if oxygen is required (sats <92%) consider wafting rather than a mask
    - nebulised adrenaline
        - only used in severe or life-threatening croup
        - 0.5ml/kg of 1:1,000 concentration to a maximum of 5ml
        - improvement occurs within 30 minutes and lasts for up to 2 hours
            - afterwards, the child returns to baseline or may deteriorate further
        - adrenaline rapidly improves distress and allows time for an appropriate team to be gathered
    - corticosteroids
        - oral dexamethasone 0.15mg/kg
        - superior to prednisolone
        - can be oral or IM
        - an alternative is 2mg nebulised bumetanide, however this can agitate the child and is more expensive so dexamethasone is preferred
        - corticosteroids take between 2 and 4 hours to have a clinical effect
    - summary of management
        - mild croup (0-2)
            - single dose of oral dexamethasone
            - parental information
            - discharge if safe

        - moderate croup (3-5)
            - dose of oral dexamethasone
            - observe for 4 hours
            - if improved:
                - parental information
                - discharge if safe
            - if partially improved:
                - observe
                - repeat corticosteroids at 12 hours
                - parental information
            - if not improved:
                - admission
                - re-assess patient
                - re-consider diagnosis
        - severe croup (6-11)
            - do not disturb child
            - dose of oral dexamethasone
            - consider adrenaline
            - consider need for intubation
            - admit
        - life-threatening croup (>11)
            - 100% oxygen and nebulised adrenaline
            - senior anaesthetist and senior help
- complications
    - some children do not respond to steroids and can deteriorate
    - after adrenaline there can be a rebound effect with rapid deterioration
    - uncommon complications include pneumonia and bacterial tracheitis
- differential for stridor
    - acute stridor
        - croup
        - foreign body aspiration
            - usually food
            - peak incidence 1-2 years
            - usual history is sudden onset retching, choking and coughing
        - angioedema
            - can be allergic, hereditary or idiopathic
            - allergic angioedema
                - may occur with or without urticaria
                - airway compromise is caused by vasodilatation and associated oedema
                - treatment is with IM adrenaline, oxygen, steroids, H1 and H2 blockers, IV fluids, consideration of intubation
                - all children given adrenaline must be admitted for observation
                - children should be referred to an allergy specialist on discharge and discharged with 2 autoinjectors (one to be kept at school) and training in how to use them

- hereditary angioedema
    - autosomal dominant disorder of C1 esterase inhibitor
    - results in release of bradykinin and C2-kinin mediators
    - this enhances vascular permeability and leads to extravascular fluid shifts
    - around 40% of people with HAE present before 5 years and 75% before 15 years
    - attacks usually occur at a single site; lifetime risk of laryngeal involvement is 70% but this is uncommon in children
    - subcutaneous angioedema is circumscribed, non-pruritic, non-erythematous swelling of the skin – almost 100% of patients with HAE have this in their lifetime
    - 45% of attacks involve the limbs – it can also develop on the face, neck, genitals and trunk
    - skin oedema occurs in 50% of attacks
    - abdominal attacks mimic acute abdomen with abdominal pain, vomiting, diarrhoea and even ileus
    - for severe HAE attacks (e.g. facial/tongue/oropharyngeal swelling, dysphagia, voice alteration or severe abdominal pain, C1 inhibitor concentrate should be administered
        - doses:
            - <50kg: 10 units/kg
            - 50-100kg: 1000 units
            - >100kg: 1500 units
        - clinical improvement is seen in 15-60 minutes
        - repeat dose may be required if there is no improvement within an hour or there is deterioration
        - if C1 inhibitor concentrate is not available, fresh frozen plasma or solvent detergent treated plasma (Octaplas) can be used
    - HAE does not respond to adrenaline
- abscess (peritonsillar or parapharyngeal)
    - origin is spread from the teeth, middle ear or sinuses
    - most common bacteria are streptococcus pyogenes, staphylococcus aureus, haemophilus influenzae, Neisseria spp and anaerobes
    - presentation is with fever, sore throat and poor oral intake
    - examination may reveal a neck mass, fever, cervical adenopathy, neck stiffness or torticollis, agitation, lethargy, drooling, trismus and stridor
    - lateral soft tissue x-rays in stable patients may show enlarged prevertebral soft tissue swelling
- epiglottitis
- bacterial tracheitis
    - may occur at any age

                - may present similarly to croup in the early stages but there is a failure to respond or only transient response to steroids and adrenaline and the condition worsens
                - the larynx, trachea and bronchi can become obstructed with purulent debris
                - an adherent pseudomembrane forms over the tracheal mucosa that can slough off, causing an obstruction
                - there is normally a preceding upper respiratory tract infection for a few days followed by rapid deterioration with fever and respiratory distress
                - there is cough, productive of copious secretions, and retrosternal pain
                - there is no dysphagia or drooling (like in epiglottitis)
                - treatment is with IV antibiotics, with intubation often needed for airway control, management of respiratory failure and pulmonary toilet
                - young children can deteriorate quickly due to the small size of the airway
                - complications:
                    - pneumonia
                    - acute respiratory distress syndrome
                    - septic shock
                    - pulmonary oedema
                    - pneumothorax
                    - cardiorespiratory arrest (rare)
        - chronic stridor
            - laryngomalacia
                - 75% of cases
                - due to a congenital abnormality of the laryngeal cartilage causing it to collapse on inspiration
                - 99% resolve spontaneously by 2 years of age
            - vocal cord dysfunction
            - subglottic stenosis
            - laryngeal webs
            - laryngeal cysts
            - laryngeal haemangioma
            - tracheomalacia
            - tracheal stenosis
- comparison between croup, epiglottitis and tracheitis
    - croup
        - incidence
            - common
        - age
            - 6 months to 3 years
        - aetiology
            - viral
        - speed of onset

        - slow
    - fever
        - rarely >39 degrees
    - cough
        - barking
    - voice
        - hoarse
    - position
        - supine
    - neck x-ray AP
        - steeple sign
    - neck x-ray lateral
        - normal
    - response to adrenaline
        - very good
- epiglottitis
    - incidence
        - rare
    - age
        - 2-7 years
    - aetiology
        - bacterial
    - speed of onset
        - very rapid
    - fever
        - normally >39 degrees
    - cough
        - suppressed
    - voice
        - muffled
    - position
        - sitting forward, neck extended
    - neck x-ray AP
        - normal
    - neck x-ray lateral
        - thumb print
    - response to adrenaline
        - no response
- tracheitis
    - incidence
        - rare
    - age
        - 6 months – 14 years
    - aetiology
        - bacterial
    - speed of onset
        - rapid

        - fever
            - normally >39 degrees
        - cough
            - present
        - voice
            - hoarse
        - position
            - supine
        - neck x-ray AP
            - steeple sign
        - neck x-ray lateral
            - hazy
        - response to adrenaline
            - partial or no response

EC2 epiglottitis

- rare, becoming more common in adults
    - less common in children since introduction of Haemophilus Influenza type B (Hib) vaccine
    - 2-5 years in children, 40s and 50s in adults
    - more common in men
    - 1-4/100,000 incidence
- suspect in patients with sore throat and a normal-looking oropharynx
- causes
    - bacterial
        - Streptococcus spp
        - Staph aureus
        - H. influenzae type B
        - Pseudomonas
        - Moxarella catarrhalis
        - Myhcobacterium tuberculosis
    - viral
        - herpes simplex
    - fungal
        - Candida spp
        - Aspergillus spp
    - non-infectious
        - thermal (steam, crack cocaine)
        - caustic (dishwasher tabs)
        - foreign bodies
        - trauma
- signs and symptoms (may decompensate rapidly)
    - sore throat
    - odynophagia
    - drooling/spitting
    - muffled voice
    - fever

    - pyrexia
    - tachycardia
    - anterior neck tenderness over hyoid
    - ear pain
    - cervical lymphadenopathy
    - tripod posture
    - in more severe cases:
        - dyspnoea
        - dysphagia
        - dysphonia
        - respiratory distress
        - stridor
- impending airway disaster triad:
    - rapid onset aphagia or severe dysphagia, frequently associated with severe sore throat
    - rapid onset laryngeal voice change: hoarse, croaky, husky or no voice
    - systemically very unwell: pyrexia, tachycardia, tachypnoea
- investigation
    - fibre-optic laryngoscopy by skilled operator with anaesthetic support present
    - lateral x-ray may show thumb print sign
- management
    - keep patient calm
    - immediate anaesthetic and ENT review if stridulous/unwell
    - IV antibiotics (cephalosporin and metronidazole)
    - IV steroid (dexamethasone 8mg)
    - IV fluids
    - nebulised adrenaline to buy time if stridor present
        - 1-5mg (1mg = 1ml of 1:1000)
    - may require intubation or surgical tracheostomy

EC3 glandular fever

- 80-90% caused by EBV (human herpesvirus 4)
    - other causes include:
        - CMV
        - human herpesvirus 6
        - toxoplasmosis
        - HIV
        - adenovirus
- epidemiology
    - incidence 5/1000
    - more common in populations with young adults (e.g. college, military)
    - no gender difference or seasonal variation
- presentation
    - often asymptomatic
    - low grade fever, fatigue, malaise (may persist for several months)
    - sore throat
        - exudative massive tonsillar enlargement

        - palatal petechiae
        - uvular oedema
    - macular rash
    - transient bilateral upper lid oedema
    - lymphadenopathy, particularly neck glands
    - nausea and anorexia
    - arthralgia and myalgia
    - cough, chest pain, photophobia
    - later signs:
        - mild hepatomegaly and splenomegaly
        - jaundice
- investigations
    - if under 12 or immunocompromised:
        - EBV serology after being ill for at least 7 days
    - if older than 12:
        - FBC (shows lymphocytosis) and monospot test in 2nd week of illness
- heterophile antibodies
    - produced by 70-90% of patients after EBV infectious mononucleosis
        - these are antibodies produced by one species that react against antigens from another species
    - Paul-Bunnell test:
        - sheep red blood cells (agglutinate in presence of heterophile antibodies)
    - Monospot test:
        - horse red blood cells
        - sensitivity 70-90%, specificity 100%
    - tests may be negative early on
    - agglutinins normally present for 4-8 weeks
- management
    - avoid contact sports for 3 weeks (consider US abdomen)
    - avoid alcohol
    - paracetamol
    - avoid amoxicillin (causes itchy rash)
    - last 2-4 weeks
        - fatigue can persist
        - can reactivate in immunocompromise
- complications
    - upper airway obstruction
    - myocarditis
    - splenic rupture
    - haemolytic anaemia

EC4 LMN facial nerve palsy

- facial nerve course:
    - originates from its nucleus in the pons
    - travels past the cerebello-pontine angle through the petrous temporal bone
        - with the chorda tympani
    - emerges from the stylomastoid foramen

    - passes through the parotid gland
        - divides here
- presentation
    - weakness of entire half of face (no forehead sparing)
- causes
    - Bell's palsy (most common)
    - pontine tumour
    - acoustic neuroma at cerebello-pontine angle
    - Ramsay-Hunt syndrome
    - trauma (base of skull or facial fracture)
    - middle ear infection/cholesteatoma
    - sarcoidosis
    - parotid gland tumour
- Bell's palsy
    - unclear cause
    - more common in pregnancy and diabetes
    - may be related to viral infection causing swelling of the nerve within the temporal bone
    - may have loss of taste on anterior two thirds of tongue (chorda tympani) and hyperacusis (stapedius)
    - if presenting within first 72 hours:
        - prednisolone 25mg BD for 10m days
        - no evidence of benefit after 72 hours
    - prescribe lubricant eye drops and advise taping of eyelid at night to avoid corneal abrasion
    - 85% have full recovery within 9 months
- Ramsay-Hunt syndrome
    - herpes zoster infection of geniculate ganglion
    - vesicles in external auditory meatus
    - treated with acyclovir and prednisolone (although not shown to be better than prednisolone alone)
    - refer to ENT
- House-Brackmann facial nerve palsy grading system
    - I (normal)
    - II (slight)
        - slight weakness on close inspection
        - slight synkinesis (abnormal facial nerve regeneration causing abnormal synchronisation and muscles other than those intended to contract)
    - III (moderate)
        - obvious but not disfiguring facial asymmetry
        - synkinesis noticeable but not severe
        - may have hemifacial spasm or contracture
        - complete eyelid closure with effort
        - mouth slightly weak with maximum effort
    - IV (moderately severe)
        - disfiguring facial asymmetry and/or obvious facial weakness
        - forehead cannot move
        - incomplete eyelid closure

        - mouth asymmetric with maximum effort
    - V (severe)
        - only slight, barely noticeable, facial movement
        - asymmetric facial appearance
        - forehead cannot move
        - incomplete eyelid closure
        - mouth has only slight movement
    - VI (total)
        - no facial function

EC5 Ménière's disease

- cause
    - distension of the membranous labyrinth due to too much endolymph
    - causes vertigo if it injures the vestibular system or hearing loss if it injures the cochlea
    - exact cause is unknown
        - possible risk factors include allergy, autoimmunity, genetic susceptibility, metabolic disturbance, vascular factors, viral infection
    - incidence around 13/100,000
    - common age 40-50
    - has been reported in children
- presentation
    - symptoms:
        - vertigo
        - tinnitus
        - fluctuating hearing loss with sensation of aural pressure
    - attacks last minutes to hours (2-3 hours)
    - may occur in clusters
    - usually unilateral but may become bilateral
    - 4% have 'drop attacks' – sudden falls without loss of consciousness or vertigo
    - definitive diagnosis is by ENT
- patients with vertigo must send a form to the DVLA
- management
    - acute attacks
        - prochlorperazine, cinnarizine, cyclizine or promethazine for vertigo and nausea
            - buccal or IM if vomiting
        - may need admission for severe attacks
        - IM steroid injection followed by tapering oral dose may help
    - prophylactic measures
        - low salt diet, avoidance of caffeine, chocolate, alcohol and tobacco
        - betahistine 16mg TDS

EC6 nasal fractures

- most common facial fracture
- clinical diagnosis
- complications include:

    - septal haematoma
        - blood collecting under lining of septal cartilage causing red/purple fluctuant swelling on both sides of the nasal septum
        - check other side for concavity which would imply deviated cartilage rather than haematoma
        - can palpate (e.g. with Jobson Horne probe) for fluctuance
        - can also be confirmed by aspirating blood/clot with green needle
    - CSF leak
        - unlikely in isolated nasal injury with no loss of consciousness
        - continuous clear and watery discharge
        - usually attend after a few days
        - usually unilateral
        - send a few ml for beta-2 transferrin/tau protein
        - patient can catch sample at home if needed
        - manage conservatively if no meningism/neurology
            - no benefit from antibiotic prophylaxis
            - most heal spontaneously
    - anosmia
    - septal deviation leading to nasal obstruction
- always assess for septal haematoma
    - refer urgently for incision and drainage
    - complications if untreated include:
        - septal abscess
        - ascending cavernous sinus infection
        - necrosis of septal cartilage and saddle deformity and/or septal perforation
- arrange ENT clinic follow up at 5-7 days once swelling had subsided in case manipulation required
    - MUA should be within 7-14 days of injury, definitely before 21 days
    - unlikely to improve nasal obstruction

EC7 otitis externa

- infection of the external auditory canal ('swimmer's ear')
- usually occurs after change in environment of ear canal
    - increased humidity
    - change in pH due to impacted wax or no wax
    - break in skin
        - eczema, cotton bud trauma, foreign body
- usually due to Pseudomonas, Staph. aureus or Strep. pneumoniae
- presentation
    - pain
    - pruritus
    - discharge
        - custard-like
    - hearing loss
    - tinnitus
- examination
    - inflamed oedematous external canal

        - may contain debris obscuring the tympanic membrane
    - palpation over tragus and movement of pinna is uncomfortable
- management
    - keep ear dry (use cotton wool with Vaseline over when showering
    - do not insert anything into ear
    - topical ear drops (combined corticosteroid and antibiotic)
        - aminoglycosides contraindicated if perforation present
    - aural toilet +/- wick insertion if extensive debris
    - IV antibiotics and urgent ENT referral if concerns about malignant otitis externa
- red flags
    - complete acute stenosis of the ear canal
    - cellulitis of pinna or peri-auricular area
    - ipsilateral cranial nerve palsy
    - ipsilateral severe deep otalgia (e.g. causing insomnia)
- EAR Score for referral
- significant risk factors
        - one of: age >65, recurrent AOE, current chemo- or radiotherapy, diabetes mellitus (well-controlled)
            - score 1
        - either immune compromise (e.g. HIV, transplant) or diabetes (poorly-controlled)
            - score 2
- length of treatment
        - either unplanned re-presentation in first 10 days of treatment or AOE not resolving for more than 14 days despite treatment
            - score 3
- red flags
        - one of: cranial nerve palsy, disproportionate ipsilateral head pain, erythema and swelling of pinna or face, completely stenosed ear canal (unable to insert speculum at all)
            - score 5
    - 0= unlikely to require specialist referral now
    - 1-2= active monitoring – review in primary care during and after treatment
    - 3-4= consider specialist review within 12-48 hours
    - 5+ or any red flag= urgent specialist referral
- Necrotising otitis externa (NOE)
    - osteomyelitis of temporal bone and skull base
    - most commonly pseudomonas aeruginosa
    - all immunocompromised patients at risk; classical presentation is older male patient with diabetes
    - mortality up to 15% when treated, 75% with delayed diagnosis and treatment
        - facial nerve palsy poor prognostic factor
    - presentation
        - non-resolving AOE despite topical treatment
        - deep severe pain out of proportion (can cause insomnia)
        - purulent otorrhoea
        - granulation and necrotic tissue within ear canal

        - more severe cases can have conductive hearing loss and lower cranial nerve neuropathies (VII, IX, X, XI, XII) due to spread along skull base
            - may become life threatening if extending to jugular foramen
        - can rarely involve petrous apex causing V and VI nerve palsies (Gradenigo's syndrome)
    - management
        - CT temporal bone
        - antibiotics IV (usually ceftazidine or tazocin with oral ciprofloxacin (check local guidelines)
            - often prolonged courses (6 weeks)
        - tight blood sugar control for diabetics
        - surgical debridement may be required
        - hyperbaric oxygen for refractory or recurrent cases with extensive skull base and intracranial involvement

EC8 otitis media

- infection of middle ear
- commonly bacterial: Strep. pneumoniae and Haemophilus influenzae or viral: respiratory syncytial virus or rhinovirus
- 75% in under 10s
- natural history 4 days
- presentation
    - earache and deafness
    - non-specific symptoms
        - fever, lethargy, irritability, poor feeding
    - tympanic membrane red, inflamed, loss of light reflex
        - may be perforated with purulent discharge in external canal
- document facial nerve examination, neurological status, ear examination including mastoid area
- management
    - symptomatic relief
        - simple analgesia
        - antipyretics
    - avoid antibiotics
        - reduce number of days of pain slightly but not length of illness or outcome
        - consider delayed prescription
        - consider immediate antibiotic prescription for:
            - bilateral otitis media in children <2
            - acute otitis media in children with otorrhoea
- red flags
    - sepsis with post auricular swelling
    - cranial nerve palsy
    - symptoms of meningism
    - altered conscious state
- patients with red flags may need CT +/- LP
- complications
    - extracranial

        - facial palsy
            - no sparing of frontalis
            - record grade on House-Brackmann scale
            - usually due to dehiscent facial nerve canal so infection affects nerve
            - usually recovers when infection resolved
        - mastoiditis
            - abscess in mastoid air spaces of temporal bone
            - tenderness of mastoid can be normal if patient systemically well
            - patients with mastoiditis are septic with pyrexia, anorexia, lethargy
            - signs and symptoms of underlying ear infection – red bulging tympanic membrane or purulent discharge
            - loss of auriculomastoid sulcus (sharp angle between ear and mastoid)
            - pinna pushed downwards and forwards with boggy oedema of mastoid
            - can spread to other areas
        - petrositis
            - infection spreading to apex of petrous temporal bone
            - sepsis and signs and symptoms of mastoiditis
            - triad of symptoms (Gradenigo's Syndrome)
                - purulent otorrhoea
                - pain deep inside ear and eye
                - ipsilateral lateral rectus palsy
    - intracranial complications
        - meningitis
        - sigmoid sinus thrombosis
            - sepsis, swinging pyrexia and meningitis
            - palpable cord in neck with distal propagation of clot
            - with propagation to cavernous sinus: proptosis, ophthalmoplegia, chemosis
        - brain abscess
            - extradural, subdural or intracerebral collections

EC9 pharyngitis

- presentation
    - hoarse voice
    - mild cough
    - fever
    - headache
    - nausea
    - tiredness
    - cervical lymphadenopathy
    - odynophagia
- usually worsens over 2-3 days, lasts around a week
    - lasts longer than a week in 1 in 10 cases
- retropharyngeal abscess
    - presentation

        - severe sore throat
        - dysphagia
        - trismus
        - stridor
        - dribbling
        - pyrexia
        - stiff neck with head tilted to one side
        - smooth bulge on one side of midline of posterior pharyngeal wall
    - management
        - anaesthetic and ENT review
        - CT neck with contrast if possible (airway not at risk)
        - usually requires drainage under anaesthetic
        - high dose IV antibiotics

EC10 post-tonsillectomy bleed

- incidence 5%
- reactionary (within 24h) or delayed (up to 14-28 days, usually 5-9 days, associated with sloughing of the eschar, loosened vessel ties or infection)
- usually venous
- smaller herald bleed can occur first
- risk factors:
    - increasing age
    - male gender
    - surgical technique (less risk if diathermy not used)
    - undetected coagulopathy
- presentation:
    - spitting blood
    - difficulty swallowing
    - pain on swallowing
    - may be occult in children as they swallow the blood
- management:
    - ABC – ensure clear airway
    - IV access, FBC, clotting, G&S
    - fluid resuscitate if needed
    - gentle suctioning of blood from mouth
    - ice pack on back of neck
    - IV TXA (no specific evidence for post-tonsillectomy bleeds but good evidence for surgical procedures in general)
    - gargle of dilute hydrogen peroxide in water 1:1 (i.e. 3% solution with 3 parts water)
    - in adults, press cotton wool pledget soaked in adrenaline (1:10,000) against the tonsillar fossa with Magill's forceps and tail of gauze outside mouth; tilt head to side or forwards
    - consider silver nitrate cautery
    - IV co-amoxiclav
    - ENT review

EC11 tonsillitis

- causes
    - viral:
        - Epstein-Barr virus
        - herpes simplex virus
        - adenoviruses
    - bacterial
        - group A β-haemolytic streptococcus
        - mycoplasma
        - Corynebacterium diphtheriae
- clinical features
    - sore throat
    - fever
    - headache
    - mild dysphagia
    - enlarged, inflamed tonsils, possibly with suppurative exudate
    - can get referred otalgia (tympanic branch of glossopharyngeal nerve
- complications
    - otitis media
    - peri-tonsillar abscess
    - retropharyngeal abscess
    - sinusitis
    - systemic (mainly relating to group A β-haemolytic strep)
        - scarlet fever
        - rheumatic fever
        - post-streptococcal glomerulonephritis
        - infectious mononucleosis (secondary to EBV)
- investigations
    - none
    - consider throat swab and anti-streptolysin O titre in severe infection
    - Monospot or Paul-Bunnell test in suspected EBV
- management
    - symptom control
    - if unable to swallow, consider good analgesia, IV fluids and steroid
        - reassess after a few hours, may be suitable for home with analgesia and antibiotics
    - admit patients who have stertor (snoring sound of pharyngeal obstruction) – risk of hypoxia, or those still unable to tolerate oral fluids
- FeverPAIN score
    - **F**ever (during previous 24 hours)
    - **P**urulence (pus on tonsils)
    - **A**ttend rapidly (within 3 days after onset of symptoms)
    - severely **I**nflamed tonsils
    - **N**o cough or coryza
        - 1 point for each, 2-3 = 34-40% likelihood of isolating strep, 4-5 = 62-65% likelihood
- Centor criteria

    - tonsillar exudate
    - tender anterior cervical lymphadenopathy or lymphadenitis
    - history of fever (>38°)
    - absence of cough
        - score of 3-4 = 32-56% likelihood of isolating strep
- NICE guideline NG84: sore throat (acute): antimicrobial prescribing
    - use FeverPAIN or Centor criteria for consideration of antibiotics
    - advise symptoms can last for a week
    - self-care advice and safety netting
    - FeverPAIN score 2 or 3:
        - consider no antibiotic prescription with advice or back-up prescribing
            - antibiotics on average shorten symptoms by 16 hours and risk adverse effects
    - FeverPAIN 4 or 5, Centor 3 or 4:
        - consider immediate or back up antibiotic prescription
    - systemically unwell, symptoms of more serious illness, high risk of complications:
        - immediate antibiotic prescription
    - antibiotic choice in adults (for children see BNF for dosing):
        - 1st line: phenoxymethylpenicillin 500mg QDS or 1g BD for 5-10 days
        - alternatives:
            - clarithromycin 250-500mg BD for 5 days
            - erythromycin 250-500mg QDS or 500mg-1g BD for 5 days

EC12 tracheostomy emergencies

- immediate complications
    - tube dysfunction
        - cuff herniation
        - equipment failure
        - incorrect size
    - malposition
        - pretracheal dilatation and placement
        - endobronchial placement
        - occlusion of tip by carina or tracheal wall
        - transfixed trachea with oesophageal placement
    - damage to local structures
        - cricoid cartilage damage
        - tracheal laceration
        - haemorrhage
        - haematoma causing local compression
        - nerve injury
        - vascular injury
        - thyroid injury
    - air-related complications
        - surgical emphysema
        - pneumothorax
        - air embolism
        - pneumomediastinum

    - death
- delayed complications
    - infection
        - tracheostomy site, larynx, tracheobronchial tree, mediastinum
    - tracheostomy tube migration and displacement
        - accidental decannulation
        - twisting of tube
        - protraction/retraction
    - ulceration
        - mucosal
        - tracheo-innominate fistula
        - tracheo-oesophageal fistula
    - mechanical complications
        - obstruction with secretions
        - dysphagia due to mechanical compression of oesophagus
- late complications
    - bleeding
        - tracheal granuloma
        - trachea-innominate fistula
    - trachea
        - tracheal or laryngeal stenosis
        - persistent sinus at tracheostomy site
        - tracheomalacia
        - tracheal dilatation
    - other
        - aphonia/dysphonia
        - scar and cosmetic effects
        - psychological effects
- management of respiratory distress
    - DOPES
        - **d**isplacement of tube
        - **o**bstruction of tube
        - **p**atient – especially pneumothorax, also pulmonary embolism, pulmonary oedema, collapse, bronchospasm
        - **e**quipment – ventilator problems
        - '**s**tacked breaths'
    - MASH
        - **m**ovement of chest during ventilation
            - absent?
            - unilateral?
            - hyper-expanded?
        - **a**rterial saturation
        - **s**kin colour
            - turning blue or pinking up?
        - **h**aemodynamic stability
    - tube blocked or displaced?
        - requires removal or replacement

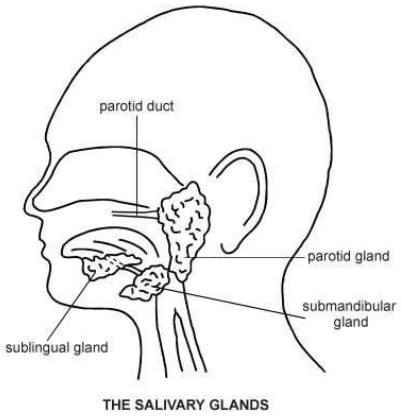
        - check if able to pass suction catheter
        - can use ETCO2, CXR and bronchoscopy as adjuncts
    - cuffed or cuffless tube?
        - if pilot balloon present it is cuffed
        - cuff leak may be present
            - cut inflation line and insert 20G cannula with 3 way stopcock as temporary one way valve to reinflate cuff if defect in distal inflation line or pilot balloon
    - what is the outer diameter of the tube?
        - use same or smaller outer diameter
            - not same as 'size'
    - when was tracheostomy performed?
        - only safe to reinsert if > 7 days
    - what was the indication?
        - if laryngectomy or supraglottic pathology intubation from above will not be possible
- management
    - assess ABCs
    - call for help
    - high flow oxygen
        - to mouth/nose and stoma site
            - only discount mouth/nose if certain there was laryngectomy
    - remove inner cannula (and plug/speaking valve if present) and check if tube is patent or displaced
        - pass suction catheter down and suction
        - if unable to pass tube is blocked or displaced
        - if able to pass could have partial blockage or displacement
        - some inner cannulae need to be replaced to connect to breathing circuits
    - if able to pass suction catheter/patency confirmed, attempt oxygenation via tracheostomy
        - seek and treat other causes of respiratory distress
        - ensure cuff inflated if positive pressure ventilation required (or replace cuffless with cuffed tube)
            - call ENT if <7 days since procedure
    - if blocked or displaced
        - remove tracheostomy tube
        - attempt oxygenation and ventilation via mouth
            - cover stoma with gauze to prevent gas escape
        - if unsuccessful attempt via stoma – use paediatric mask or LMA held over stoma
        - if unsuccessful attempt endotracheal intubation and ensure ETT advances beyond stoma (expect difficult airway)
        - if unsuccessful attempt intubation of stoma
            - size 6.0 ETT or small tracheostomy tube
            - options include bougie, airway exchange catheter, fibreoptic bronchoscopy

EC13 quinsy

- presentation
    - high fever
    - unilateral throat pain
    - increasing dysphagia
    - trismus
    - 'thick' or 'hot potato' voice (not hoarse)
    - anterior arch pushed medially on affected side
    - uvula may be pushed away
- Liverpool Peritonsillar Abscess Score (cut off value 4, likelihood increases with scores 4-8)
    - unilateral sore throat (rated 80:20 or more by patient): 3
    - trismus (inter-incisor distance <3cm): 2
    - male gender: 1
    - pharyngeal voice change (hot potato voice): 1
    - uvular deviation (away from affected side): 1
- management
    - inspect oropharynx with caution – potential for airway obstruction
    - consider epiglottitis in differential particularly if throat looks normal
    - urgent anaesthetic and senior ENT review if stridor, unable to swallow, tripod position
    - if no airway compromise, IV benzylpenicillin and referral to ENT for aspiration and drainage
    - FBC, U&E, LFT, glandular fever screen
    - analgesia (including topical analgesic spray)
    - IV fluids
    - consider 6.6mg dexamethasone, particularly if stertor present
    - aspiration by ENT
        - may need to be delayed if significant trismus – need IV fluids, steroids, analgesia first
        - patient is positioned upright with head back and neck slightly extended
        - 2-3 sprays of Xylocaine spray onto quinsy
        - uses the needle from a cannula or a large bore hypodermic needle with tape wrapped around to reduce insertable length
        - aspiration point 1 is a line traced from the medial surface of the molars until it meets a line traced horizontally from the base of the uvula
        - there is a risk of damage to the carotid artery, which is lateral
        - pus is sent for microbiology
        - oropharynx is re-examined after a rest and after 24 hours
        - incision and drainage may be needed if there is reaccumulation
    - alternative aspiration technique
        - use a laryngoscope to hold down the tongue and provide lighting
        - use a syringe with a spinal needle with the plastic cover cut off to provide a safety stop (needle pokes out of end of safety cap)
        - use local anaesthetic spray and drain abscess
        - https://coreem.net/procedures/drainage-of-a-peritonsillar-abscess/

EC14 salivary gland disease

- anatomy and physiology



THE SALIVARY GLANDS

https://patient.info/doctor/salivary-gland-disorders

  - around 1-1.5 litres/day of saliva is produced by three pairs of major salivary glands:
    - the parotid glands
      - lie below the external auditory meatus between the vertical ramus of the mandible and the mastoid process
      - the parotid duct crosses the masseter and opens via a small papilla on the buccal membrane opposite the crown of the second molar
      - the facial nerve subdivides into its branches as it passes through the parotid
    - the submandibular glands
      - walnut-sized paired structures lying beneath and in front of the angle of the jaw, wrapping around the posterior edge of the mylohyoid muscle
      - the ducts emerge to the floor of the mouth just lateral to the frenulum of the tongue
    - the sublingual glands
      - lie below the tongue and open through several ducts to the floor of the mouth
  - there are a large number of minor salivary glands (600-1000) widely distributed throughout the oral mucosa, palate, uvula, floor of the mouth, posterior tongue, retromolar and peritonsillar area, pharynx, larynx and paranasal sinuses
  - saliva is made up of:
    - water
    - electrolytes
    - lubricants
    - antimicrobial compounds
    - enzymes
    - growth factors
  - the components facilitate speech, mastication and swallowing and start the digestive process
    - saliva also prevents oral problems by protecting the oral mucosa and teeth
- history

    - which gland is affected – most commonly the parotid
    - if there is swelling, whether it is:
        - unilateral or bilateral
        - constant or intermittent
        - painful – may be referred to ear or throat
    - how long they have noticed symptoms for and whether any mass has increased in size since it was first noticed
    - whether symptoms are affected by eating
    - whether there is a feeling of dry mouth
    - whether there are systemic symptoms suggestive of infection, autoimmune disease, sarcoidosis or malignancy
    - whether there is anything of relevance in the medical or dental history or medication and immunisation record
- signs
    - examination of the major glands
        - parotid glands
            - swellings are apparent as a loss of the angle of the jaw
                - the accessory lobe may cause a lump anterior to the ear
                - the deep lobe needs to be inspected and palpated through the mouth
                - swelling can displace the ipsilateral tonsil
                - try to differentiate between generalised swelling of the gland (usually due to obstruction of the duct or inflammatory disease) or localised lumps (more likely to be tumours)
            - ask the patient to clench their teeth to allow palpation of the masseter
                - the anterior part of the parotid duct can be felt as it crosses the anterior border of the masseter muscle and occasionally a stone can be palpated in that part of the duct
                - inspect the orifice of the duct inside the mouth opposite the second upper molar by retracting the cheek with a spatula
                - pressure on the body of the gland may lead to extrusion of pus at the orifice in patients with parotitis
            - examine the facial nerve – facial weakness or asymmetry is highly suggestive of malignancy
        - submandibular glands
            - usually involves swelling beneath and anterior to the angle of the jaw
            - inspect the orifices of the duct by asking the patient to lift their tongue to the roof of the mouth
                - note the presence of inflammation, pus, or a visible impacted stone
            - examine bimanually with the index finger of one hand inside the mouth and the fingers of the other hand over the outer surface of the lump in the neck

        - the gland is not normally palpable but if enlarged can be felt 2-3cm anterior to the sternomastoid, below the horizontal ramus of the mandible
        - the gland has a rubbery consistency
        - the gland should not be fixed to the floor of the mouth or tongue
        - check the course of the duct for a stone
    - sublingual gland pathology may cause swelling on the floor of the mouth
  - establish the following:
    - is the swelling a salivary gland? it can be difficult to differentiate a swollen parotid gland and cervical lymphadenopathy
      - it is usually possible to feel in front of lymph nodes, but not the parotid
    - whether there are signs of systemic illness such as malaise or pyrexia
    - whether the eyes are dry
      - look for keratoconjunctivitis sicca and other features of Sjögren's such as xerostomia and lingual papillary atrophy
    - whether tooth enamel has been lost (e.g. recurrent vomiting with bulimia)
    - regional lymphadenopathy
- causes of salivary gland swelling
  - parotid
    - viral parotitis – mumps
    - stone in salivary duct
    - benign and malignant tumours
    - Sjögren's syndrome
    - sarcoidosis
      - Heerfordt's syndrome: sarcoidosis with parotid enlargement, fever, anterior uveitis, facial nerve palsy
    - acute and chronic bacterial parotitis
    - granulomatosis with polyangiitis
    - HIV-related lymphocytic infiltration
  - submandibular
    - stone in the salivary duct
    - benign and malignant tumours
    - Sjögren's syndrome (less common)
  - in the minor salivary glands
    - mucoceles
    - benign and malignant tumours
- infection
  - background
    - mumps is the most common cause
      - incidence has fallen with widespread immunisation
      - usually causes bilateral swelling of the parotid glands
        - may be unilateral and the other salivary glands may be affected
      - swelling lasts around a week accompanied by low grade pyrexia and general malaise

        - other viruses include Coxsackievirus, parainfluenza, influenza A, parvovirus B19, herpes
        - acute bacterial infection of the major salivary glands usually occurs in debilitated or dehydrated patients
            - comorbidity and/or medication may inhibit saliva production, increasing vulnerability
            - infection ascends from the oral cavity, usually Staphylococcus aureus
        - chronic bacterial infection may occur on a background of a gland previously damaged by stones, irradiation or autoimmune disease
            - chronic infection destroys the glandular elements and can impair the protective functions of saliva, leading to dental infections and disease – often first presents to a dentist
        - recurrent parotitis of childhood presents with swelling, pain and fever with cause unknown
        - parotid swelling may be an initial presentation of HIV – oral mucoceles and ranulas may also occur
        - TB is a rare cause of parotitis and other salivary gland swelling
    - presentation
        - swelling which is usually painful and tender
        - dry mouth
        - abnormal or foul tastes associated with purulent discharge from the salivary duct opening in bacterial infection
        - mouth or facial pain, especially associated with eating
        - decreased mouth opening, difficulty talking
        - fever, systemically unwell
    - investigation
        - may include:
            - bloods
                - FBC, inflammatory markers, U&Es, blood cultures, viral serology or salivary antibody testing, HIV testing as appropriate
            - pus swab for culture and sensitivities (massage gland to express if needed)
            - sialography
            - ultrasound
            - CT/MRI – often to exclude neoplasm
            - fine needle aspirate or incisional biopsy for histology or culture material where relevant
    - management
        - mumps is self-limiting without serious sequelae in most patients
            - supportive treatment is appropriate
            - it is a notifiable disease
        - acute suppurative infection is treated with antibiotics and incision and drainage if an abscess has developed
        - salivary flow should be encouraged:
            - warm compresses

                - sialagogues such as lemon drops, gum, vitamin C lozenges
                - hydration
                - salivary gland massage
                - oral hygiene
            - with chronic infections, if duct obstruction is identified, stones or strictures can be removed to improve salivary flow
                - gland excision may be required for recurrent problems
    - complications
        - complications of mumps parotitis include: orchitis, oophoritis, aseptic meningitis, deafness; rarely sialectasia and recurrent sialadenitis
        - abscess formation with spread to the other deep spaces of the neck
            - trismus may indicate parapharyngeal involvement
            - Ludwig's angina with infection of the submental and sublingual spaces
- obstruction
    - background
        - calculi can form in the major salivary glands and their ducts, causing obstruction of salivary outflow, typically with pain and swelling at mealtimes
            - most commonly in the submandibular gland and its duct (80-90%) but may be seen in the parotid glands
        - obstruction of minor salivary glands can also occur, resulting in cyst-like swellings in the lips and cheeks
        - the cause of salivary stones is unknown
            - they are composed of mucus, cellular debris, calcium and magnesium
        - parotid gland obstructions are more usually due to stenosis of the opening of the duct rather than stones
            - can be secondary to chronic trauma such as ill-fitting dentures
        - obstruction of a salivary duct causes inflammation and swelling of a gland
            - if the obstruction is not relieved, the gland becomes damaged and may require excision
    - presentation
        - usually colicky postprandial swelling of the gland
        - symptoms typically relapse and remit
    - investigation
        - ultrasound
            - stones show as hyperechoic lines or points with distal acoustic shadowing
            - can exclude other causes such as malignancy and lymphadenopathy
        - contrast sialography
            - provides information about the ductal system
            - obstruction is indicated by filling defects or strictures
        - CT scan
    - management
        - many stones pass spontaneously
            - conservative treatment of analgesics
            - oral antibiotics where there is evidence of infection

                - good hydration, warm compresses and gland massage to assist passage of stones
            - surgical management
                - proximal submandibular stones may be removed by dilating/incising Wharton's duct and a transoral approach
                - calculi in the submandibular duct may be removed by an incision in the floor of the mouth, those in the substance of the gland may require gland excision
                - endoscopic or minimally invasive techniques or lithotripsy are used where possible with the aim of better preserved gland function
- sialadenosis
    - background
        - generalised gland swelling caused by hypertrophy of the acinar component of the gland
        - associated with a number of systemic diseases
        - treatment is aimed at the underlying cause
        - the most common cause is Sjögren's syndrome
            - preferentially affects the parotids but may also affect the submandibular and minor salivary glands; also usually affects the lacrimal glands
            - Sjögren's may be accompanied by other systemic diseases such as rheumatoid arthritis, SLE or primary biliary sclerosis
            - strong female to male predominance (9:1), onset typically in middle age
    - other causes of sialadenosis
        - bulimia
        - anorexia
        - endocrine disorders such as Cushing's syndrome, diabetes and hypothyroidism
        - coeliac disease and malnutrition
        - alcoholism
        - drug induced, e.g. anticholinergics
        - sarcoidosis and Heerfordt's syndrome
    - investigations include
        - sialogram
        - biopsy of the labial salivary glands
        - autoantibodies for Sjögren's
        - rheumatoid factor (positive in about 90%)
        - antinuclear antibodies
        - CXR if sarcoidosis suspected
        - other investigations as indicated
    - management
        - Sjögren's requires referral to rheumatology
        - good dental care is essential to prevent caries
        - parasympathetic drugs such as pilocarpine may be used to treat hyposalivation and xerostomia

        - there is inadequate evidence to recommend local stimulants, lubricants and protectants, although they are widely used
        - gland excision is rarely indicated
    - complications
        - risk of malignant non-Hodgkin's lymphoma is increased in primary Sjögren's syndrome
            - may be difficult to diagnose in the context of persistent parotid swelling
- tumours
    - background
        - malignant tumours of the salivary glands are rare, most neoplasms are benign
        - all salivary swellings require urgent referral and investigation
    - red flags
        - rapid increase in size of swelling
        - ulceration and/or induration of the mucosa or skin
        - fixation to the skin
        - paraesthesia/anaesthesia of associated sensory nerves
        - past history of skin cancer, Sjögren's syndrome or radiation to the head and neck
    - investigation
        - ultrasound is first line, usually combined with fine needle aspiration for cytology or core needle biopsy where a tumour is seen
        - MRI gives more information about tumour margins and staging
        - CT and PET-CT are used to determine metastatic spread
    - management
        - tumours generally require surgical excision
        - postoperative radiotherapy may be required for malignant tumours

EC15 vestibular neuritis

- background
    - most likely a vestibular neuropathy caused by reactivation of latent type 1 herpes simplex virus in the vestibular ganglion
    - autoimmune and microvascular ischaemic insults are also possible mechanisms
    - most commonly affects the superior division of the nerve, which is much longer than the inferior division and travels through a narrow bony passage, making it more vulnerable to the effects of swelling or ischaemia
    - a prior upper respiratory tract infection is reported in up to 100% of cases
    - affects adults and children
        - peak onset 40-50 years
        - incidence around 3.5 cases per 100,000
- history
    - sudden, spontaneous, severe and often incapacitating vertigo
        - vertigo (the illusion of movement) is constant and ongoing
        - it is not triggered by movement but may be exacerbated by movement
    - nausea and vomiting are frequent
    - there is never hearing loss

    - preceding or concurrent upper respiratory tract symptoms
        - there may be fever, however high fever suggests a more serious cause such as mastoiditis or meningitis
    - 25% of cases have a brief prodrome in the week before the attack; more than one suggests TIA
    - drugs that may cause acute vertigo:
        - aminoglycosides and other ototoxic medications
        - antihypertensives such as amlodipine
        - antidepressants
            - abrupt discontinuation of an SSRI may cause vertigo
        - tranquilisers, including benzodiazepines
        - anti-epileptics
    - carbon monoxide exposure is a rare cause of acute vertigo
- examination
    - assessment of the external ear and tympanic membrane
        - looking for cholesteatoma or vesicles suggestive of herpes zoster
    - cranial nerve examination
        - for evidence of palsies and hearing loss
    - mastoid tenderness
    - nuchal rigidity
    - high fever
    - assessment of gait
        - patients tend to fall towards the affected side when standing or walking
        - they should still be able to sit and stand unaided (the brain can still process information from the visual and somatosensory systems)
            - inability to stand or walk unassisted may suggest ischaemia
    - hearing test
        - using 512 Hz tuning fork
        - Weber's test
            - tuning fork on top of forehead and ask which ear it is louder in
                - should be no difference
                - in sensorineural loss it is quieter in the affected ear
                - in conductive loss it is transmitted through the skull and louder in the affected ear
    - HiNTS test
- investigations
    - bloods
        - not helpful unless systemic infection suspected, where FBC and cultures should be done
    - culture and sensitivity of middle ear effusions if present
    - imaging
        - not normally required
        - CT can help rule out mastoiditis
        - temporal bone CT can help with cholesteatoma and labyrinthitis
        - MRI is more useful if a sinister cause is suspected
            - still has a low sensitivity for ischaemic stroke in dizziness, particularly in the first 24-48 hours

- management
    - reassurance
        - symptoms resolve after several days to a few weeks with or without symptomatic relief
    - encourage the patient to be active as soon as possible, even if it worsens vertigo, as it is likely to help with vestibular compensation
    - advise to seek medical review for worsening symptoms, particularly neurological symptoms
    - medication
        - should be taken for the minimum time so as not to delay compensation and recovery
        - prochlorperazine and antihistamines may help symptoms
            - buccal or deep IM injection prochlorperazine can be considered if symptoms are severe
        - antivirals, benzodiazepines and corticosteroids are not recommended
    - vestibular rehabilitation
        - physical manoeuvres and exercise regimes
        - safe and effective and improves functioning in the medium term
    - the patient should be advised not to drive or operate machinery whilst they are experiencing symptoms of vertigo or taking medication for it

ELDERLY CARE/FRAILTY

ElP1 delirium

- background
    - common in hospital medicine, with a prevalence of 11-42% in the elderly
    - patients with delirium have:
        - increased length of hospital stay
        - higher risk of complications in medical and surgical settings
        - higher mortality in hospital and up to 6 months afterwards
    - a third of cases are thought to be preventable
    - detection and documentation of delirium by emergency physicians is poor
- clinical presentation
    - diagnosis of delirium is clinical, defined by the DSM-IV as:
        - disturbance of consciousness (i.e. reduced clarity of awareness of the environment) with reduced ability to focus, sustain or shift attention
        - a change in cognition (such as memory deficit, disorientation, language disturbance) or the development of a perceptual disturbance that is not better accounted for by a pre-existing, established or evolving dementia
        - the disturbance develops over a short period of time (usually hours to days) and tends to fluctuate during the course of the day
    - there are 3 clinical subtypes:
        - hyperactive
            - heightened arousal
            - restlessness, wandering
            - sometimes aggressive
        - hypoactive
            - decreased alertness

                - sparse/slow speech
                - lethargy, apathy
            - mixed
                - combination of the above
    - in summary, the key features of delirium are:
        - recent onset of fluctuating awareness
        - impairment of memory and attention
        - disorganised thinking
- risk factors
    - risk factors for developing delirium
        - old age
        - severe illness
        - dementia
        - physical frailty
        - admission with infection or dehydration
        - visual impairment
        - polypharmacy
        - surgery
        - alcohol excess
        - renal impairment
    - precipitating factors for delirium
        - immobility
        - use of physical restraint
        - use of bladder catheter
        - iatrogenic events
        - malnutrition
        - psychoactive medication
        - intercurrent illness
        - dehydration
    - causes or precipitants of acute
    - mnemonic: SMASHED
        - **S**ubstrates (hyperglycaemia, hypoglycaemia, thiamine)/**S**epsis
        - **M**eningitis and other CNS infections/**M**ental illness, functional psychoses
        - **A**lcohol intoxication or withdrawal
        - **S**eizures (seizure activity, post-ictal states)/**S**timulants (anticholinergics, hallucinogens, cocaine)
        - **H**yper (hyperthyroidism, hyperthermia, hypercarbia)/**H**ypo (hypothyroidism, hypothermia, hypoxia, hypotension)
        - **E**lectrolytes (hypernatraemia, hyponatraemia, hypercalcaemia)/**E**ncephalopathy (hepatic, uraemic, hypertensive, others)
        - **D**rugs of any sort
- history (with collateral where possible)
    - onset and course of delirium
    - previous intellectual function (e.g. ability to manage household affairs, pay bills, compliance with medications, use of telephone and transport)
    - full drug history (including non-prescribed drugs and recent drug cessation)
    - alcohol history
    - functional status (activities of daily living)

    - aids used (hearing aids, glasses etc)
- examination
    - full examination to look for causes of delirium
    - neurological and mental status examinations
        - Abbreviated Mental Test (AMT) and Confusion Assessment Method (CAM) are the quickest and most widely used but cannot distinguish between delirium and other causes of cognitive impairment
        - AMT (less than 8/10 is abnormal)
- age
- time (to nearest hour)
- address for recall at end of test (42 West Street)
- year
- name of hospital
- recognition of 2 people (e.g. doctor, nurse)
- date of birth
- year of first world war
- name of present monarch
- count backwards 20-1 (also tests attention)
        - CAM
- acute onset and fluctuating course AND
- inattention (e.g. 20-1 test with reduced ability to maintain or shift attention) AND EITHER
- disorganised thinking, illogical or unclear ideas OR
- alteration in consciousness (usually lethargic or stuporous)
- investigations
    - directed towards particular risk factors or possible causes from history and examination
    - consider:
        - bloods
            - FBC
            - U&E
            - LFT
            - coag
            - blood cultures
            - drug levels (e.g. theophylline, digoxin)
            - B12, folate, VDRL, autoimmune screen
            - CRP/ESR
        - oxygen saturations
        - blood glucose
        - blood gas +/- carboxyhaemoglobin
        - CXR
        - urine dip +/- culture
        - ECG
        - CT
            - no evidence for routine use in the initial workup of acute delirium
            - indicated for:
                - focal neurological signs
                - confusion after head injury/fall

        - evidence of raised intracranial pressure
    - lumbar puncture
        - if meningitis or other CNS infection suspected
- management
    - identify and treat underlying cause
        - infection is one of the most common causes
            - prompt treatment with an appropriate antibiotic and collection of samples for culture
        - biochemical abnormalities should be corrected at an appropriate rate (many are subacute)
        - parenteral thiamine if alcohol abuse or withdrawal suspected
        - review of drug history and withdrawal of drugs as appropriate
            - drugs with anticholinergic activity (particularly implicated in acute confusion) include:
                - antihistamine
                    - hydroxyzine
                    - diphenhydramine
                - antispasmodic
                    - alverine
                    - hyoscine
                - tricyclic antidepressant
                    - amitriptyline
                - benzodiazepine
                    - lorazepam
                - analgesic
                    - codeine
                - antiarrhythmic
                    - digoxin
                - diuretic
                    - furosemide
                - antiparkinsonian
                    - benztropine
                - bladder stabiliser
                    - oxybutynin
                - bronchodilator
                    - theophylline
    - preventative and nursing measures
        - strategies to implement in the ED
            - appropriate lighting levels for time of day
            - regular and repeated cues to improve personal orientation
            - use of clocks to improve orientation
            - hearing aids and spectacles in good working order
            - communication with relatives as to the cause and treatment measures for delirium
            - encourage family to visit and bring in familiar objects from home
        - medical treatment considerations
            - regular analgesia if in pain

                - optimise fluid balance to prevent dehydration
                - elimination of unexpected or irritating noise or other unfamiliar distractions (e.g. pump alarms – give fluids and medication orally if possible)
                - avoid:
                    - use of physical restraint
                    - constipation
                    - catheters where possible
                    - anticholinergic drugs
                    - unnecessary inter-/intra-ward transfers
        - drug therapy
            - should be avoided where possible in the management of delirium
            - sedation may be required in certain situations:
                - to carry out essential investigations or treatment
                - to prevent patients endangering themselves or others
                - to relieve distress in a highly agitated or hallucinating patient
            - if required, haloperidol is first line treatment
                - 0.5-1mg orally or IM
                - regular reassessment and titration up to a maximum of 5-10mg
                - side effects include extrapyramidal symptoms and it should not be used in patients with Parkinson's or Lewy Body dementia
                    - regular ECG monitoring should be done to check that the QT interval does not become more prolonged
            - for delirium due to alcohol withdrawal or patients not suitable for haloperidol, a benzodiazepine would be suitable
                - lorazepam 2mg IM/IV with regular reassessment and titration to effect
- prognosis
    - younger patients may experience mild cognitive dysfunction that lasts weeks or months
    - older people often experience persistent decline in their baseline level of functioning with loss of at least one ADL after acute delirium

ElP2 deterioration in mobility

- background
    - most common risk factors for mobility impairment are older age, low physical activity, obesity, strength or balance impairment and chronic disease such as diabetes or arthritis
    - prevalence of gait and balance disorders is 10% in those 60-69 years and more than 60% in the over 80s
    - around 30% of people 65 and over have a fall at least once a year, increasing to 50% in those 80 and over
- history
    - direct complaint of problems with walking or unsteadiness
        - clarify exactly what the patient feels, for example which aspect of walking is difficult
    - falls

        - when the last fall occurred
        - how frequent falls are
        - whether there is syncope or presyncope
    - duration of problems
    - full systems review
    - any features of cord compression
    - full drug history
- examination
    - pulse rate, rhythm and volume and presence or absence of carotid bruits
    - blood pressure including postural hypotension
    - cardiovascular examination including murmurs
    - full neurological examination
        - pyramidal, extrapyramidal and cerebellar dysfunction
        - sensation/peripheral neuropathy
    - assess for fractures and injuries
        - leg asymmetry
        - tenderness of spine/lower limbs
    - gait examination
        - asymmetrical or symmetrical problems
        - waddling gait
        - broad-based gait
        - scissoring gait (bilateral leg spasticity)
        - ataxia
    - further testing
        - Timed Up & Go test
            - time the person getting up from a chair without using their arms, walking three metres, turning around, returning to the chair and sitting down
            - usual walking aid can be used during the test
            - 12-15 seconds or more indicates high risk of falls in older people
        - Turn 180° test
            - ask the person to stand up and step around until they are facing the opposite direction
            - if they take more than 4 steps, further assessment should be considered
- causes of difficulty in walking
    - vascular
        - cardiac
            - arrhythmias
            - hypotension
            - postural hypotension
        - neurological
            - TIAs
            - CVA
            - multi-infarct dementia
    - neurological
        - pyramidal disease

                - multiple sclerosis
                - cord compression
                - motor neurone disease
                - syringomyelia
                - spinal cord tumours
                - B12 deficiency
                - syphilis
            - extrapyramidal disease
                - tardive dyskinesia
                - akathisia
                - Parkinson's disease
                - Parkinsonism (e.g. drug induced)
            - cerebellar disease
                - cerebellar tumours
                - any ataxia (e.g. Friedreich's ataxia)
                - Wernicke's encephalopathy
            - other
                - peripheral neuropathy
                - chorea
    - orthopaedic
        - painless
            - arthrodesis of hip joints
        - painful
            - arthritides (e.g. osteoarthritis, rheumatoid arthritis)
            - spinal disease (e.g. stenosis)
            - fractures
            - foot problems (e.g. corns, bunions, ill-fitting shoes)
    - balance and co-ordination
        - Alzheimer's dementia
        - labyrinthitis
        - degenerative changes in the inner ear
    - muscles
        - myopathies
    - metabolic
        - diabetes (e.g. autonomic neuropathy, foot drop)
        - thyroid disorders
    - others
        - toxins/drugs
            - anti-hypertensive medication
            - sedatives
            - antipsychotics
            - ethanol
            - anticonvulsants
        - psychological
            - loss of confidence
            - depression
- causes of 'off legs'

- background
  - ranges from unsteadiness and difficulty in walking to dizziness or lethargy
  - exact meaning should be sought
  - causes are usually acute
- causes include:
  - urine or chest infections
  - dehydration
  - neurological causes
    - head injury
    - cord compression/cauda equina syndrome
  - orthopaedic causes
    - fractures
  - metabolic abnormalities
    - hyponatraemia
    - hypercalcaemia
    - hypoglycaemia
    - hyperglycaemia
  - alcohol, drugs, medications
  - hypoxia
- investigations
  - should be guided by history and examination
  - may include cerebral imaging (CT or MRI) and blood tests (e.g. TFTs, syphilis serology)
- management
  - directed towards underlying cause
  - may need a multidisciplinary approach (e.g. physiotherapist, occupational therapist, allied healthcare professionals)

ElP3 falls

- background
  - common presentation to emergency departments
  - can be debilitating and distressing to patients
  - can be complex in aetiology and outcome
  - definition:
    - an unintentional or unexpected loss of balance resulting in coming to rest on the floor, the ground, or an object below knee level (NICE 2013)
- pathophysiology
  - patient pathology may contribute to or be caused by the fall
    - e.g. impaired consciousness, ability to balance, safety at home
  - patients with a 'long lie' (>1 hour) are at risk of complications such as dehydration, pressure sores, pneumonia, hypothermia and rhabdomyolysis
- history
  - details of what happened before and during the fall plus any witness history
  - whether the patient has their care needs met and are in a safe domestic environment
  - when?
    - before

        - whether they can recall what time they fell
    - during
        - what they were doing when they fell
            - e.g. getting up from sitting suggestive of postural hypotension
    - after
        - when they got up, how long the lie was
- what?
    - before
        - any warning signs (e.g. dizziness, chest pain)
        - systems review
            - cardiovascular
                - chest pain
                - syncope
                - palpitations
            - respiratory
                - dyspnoea
                - cough
            - neurology
                - impaired consciousness
                - seizures
                - weakness
                - slurred speech
            - gastrointestinal
                - abdominal pain
                - change in bowel habit
            - genitourinary
                - dysuria
                - urinary retention
            - general
                - reduced mobility or exercise tolerance
                - neglect (e.g. inadequate feeding, poor hygiene)
                - inability to perform ADLs
                - weight loss
    - during
        - what surface they fell on (e.g. hard floor, carpet)
        - what distance they fell from (e.g. standing height, stairs)
        - any loss of consciousness/amnesia
        - any evidence of seizure activity
        - any vertigo
    - after
        - injuries sustained
- how?
    - before
        - any changes in medication
        - reduced mobility or recent falls
        - other predisposing risk factors

        - during
            - how they think it happened
        - after
            - how they have been affected (e.g. weakness, lower limb injury)
            - whether they are confused (if so, recollection of events may not be trustworthy)
    - who?
        - before
            - whether anyone lives with the patient
            - whether there is a care package in place
        - during
            - whether anyone witnessed the fall (whether there is a collateral history available)
        - after
            - whether anyone needed to assist the patient off the floor
            - whether they need assistance in resuming ADLs
- predisposing risk factors
    - age >65 years
    - increasing frailty
        - consider consulting electronic frailty index (eFi) if available
    - cognitive/visual impairment
    - reduced mobility
        - e.g. arthritis, previous CVA, Parkinson's disease, diabetes
    - medication history
        - particularly polypharmacy
        - psychoactive drugs (e.g. benzodiazepines)
        - antihypertensives
        - diabetic medications (risk of hypoglycaemia)
        - antibiotics (suggest recent infection)
        - anticoagulants
    - environmental hazards
    - osteoporosis
    - alcohol misuse
    - depression
    - recent falls
- assessment for delirium
    - 4AT test
- alertness
- Abbreviated Mental Test (AMT-4)
    - age, date of birth, place (name of building/hospital), current year
- attention
    - list months in reverse starting from December
- acute change/fluctuating course
    - significant variation in mental status over past 2 weeks and persisting over past 24 hours
- examination (head to toe)
    - head

        - CNS
            - level of consciousness (GCS or AVPU)
            - pupils (PEARL)
            - facial weakness, slurred speech, posterior fossa signs
            - evidence of head injury (e.g. scalp laceration, Battle's sign)
        - ENT
            - haemotympanum
    - neck
        - any suspicion of c-spine injury
    - chest
        - respiratory
            - increased breathing effort
            - dullness to percussion
            - crepitations
        - cardiovascular
            - murmurs
            - pulse (arrhythmia)
            - blood pressure (septic shock, dehydration)
        - musculoskeletal
            - evidence of rib fractures
    - abdomen
        - gastrointestinal
            - abdominal tenderness
        - genitourinary
            - distended bladder (urinary retention)
    - hips
        - musculoskeletal
            - fractured neck of femur
    - limbs
        - neurological
            - weakness
            - increased tone
            - poor co-ordination
            - gait
        - musculoskeletal
            - tenderness or swelling
            - reduced mobility
                - if possible, have them walk for 3 metres with usual walking aid
    - back
        - musculoskeletal
            - spinal tenderness/bruising/swelling
- Clinical Frailty Score (Rockwood)
    - very fit
            - robust, active, energetic and motivated
            - commonly exercise regularly
            - among the fittest for their age

- well
    - no active disease symptoms but less fit than category 1
    - often they are seasonally active or exercise occasionally
- managing well
    - medical problems are well controlled
    - not regularly active beyond routine walking
- vulnerable
    - not dependent on others for daily help but symptoms often limit activities
    - common complaint is being 'slowed up' and/or being tired during the day
- mildly frail
    - often have more evident slowing and need help in higher order IADLs (finances, transportation, heavy housework, medications)
    - typically, mild frailty progressively impairs shopping and walking outside alone, meal preparation and housework
- moderately frail
    - need help with all outside activities and with keeping house
    - often have problems with stairs and need help with bathing
    - might need minimal assistance (cuing, standby) with dressing
- severely frail
    - completely dependent for personal care from whatever cause (physical or cognitive)
    - seem stable and not high risk of dying within around 6 months
- very severely frail
    - completely dependent, approaching the end of life
    - typically could not recover even from a minor illness
- terminally ill
    - approaching the end of life
    - all people with a life expectancy <6 months who are not otherwise evidently frail
- only validated in older patients (65 and over)
- collateral history can be very valuable
- if acutely unwell, they should be scored on how they were two weeks ago, not today
- history is crucial

- investigations
    - bedside
        - observations
        - blood glucose
        - cognitive assessment
        - ECG
        - urinalysis
    - serum investigations
        - FBC (evidence of infection)
        - U&Es (AKI, electrolyte abnormalities)
        - bone profile (part of confusion screen)
        - LFTs/INR (alcoholic liver disease)

        - CK
    - radiology
        - CXR
            - if concerns about pneumonia
        - CT head
            - if concerns about CVA or subdural/extradural
            - NICE guidelines for scan after head injury include:
                - loss of consciousness
                - age 65 or older
                - any history of bleeding or clotting disorders
                - dangerous mechanism of injury
                    - pedestrian or cyclist struck by motor vehicle
                    - occupant ejected from motor vehicle
                    - fall from a height of >1m or 5 stairs
                - more than 30 minutes of retrograde amnesia of events immediately before the head injury
        - FAST/CT
            - if concerns of significant trauma
- management
    - address any causes or consequences of the fall
    - early analgesia
    - hydration if there is rhabdomyolysis (with caution for fluid overload)
    - address social circumstances
        - patient may require admission rather than an unsafe discharge
    - other input:
        - care packages if assistance with ADLs required
        - physiotherapy review to support patients with unstable gait
        - occupational therapy – strategies to prevent falls including modifications to home environment
        - medication review – medications which may contribute to falls or cause problems when the patient falls (anticoagulants)

EIP4 fragility fractures

- definition
    - fractures that result from mechanical forces that would not normally result in fractures (low level trauma)
        - quantified by the WHO as forces equivalent to a fall from standing height or lower
        - vertebral fractures may occur without a fall
    - an osteoporotic fracture is a fragility fracture which has occurred as the result of osteoporosis
    - risk factors for fragility fractures include:
        - osteoporosis
        - advancing age
        - other conditions affecting bone strength (e.g. acromegaly, osteogenesis imperfecta)
        - predisposition to falls due to loss of balance or poor muscle strength

- epidemiology
    - osteoporosis causes >200,000 fragility fractures/year in the UK and costs the NHS >£1.7 billion
    - there is likely to be a doubling of osteoporotic fractures over the next 50 years due to the ageing population
    - women are more affected than men
    - more than 1 in 3 women and 1 in 5 men have one or more osteoporotic fractures in their lifetime
    - following hip fracture, around half of those discharged can no longer live independently and 20% die within a year
- aetiology
    - minor falls or minor trauma
    - vertebral fractures can follow normal activity such as bending or lifting or sneezing
- risk factors
    - reduced bone mineral density is a major risk factor
    - other risk factors include:
        - increasing age (risk partly independent of reducing BMD)
        - female gender
        - low body mass (<20 kg/m^2) and anorexia nervosa
        - parental history of hip fracture
        - past history of fragility fracture (especially hip, wrist and spine)
        - corticosteroid therapy (any dose orally for three months or more)
        - Cushing's syndrome
        - alcohol intake of 3 or more units/day
        - smoking
        - ethnicity (Caucasian men and women have higher risk)
        - other causes of abnormal bone (e.g. acromegaly, osteogenesis imperfecta)
        - falls and conditions increasing the risk of falls, such as:
            - visual impairment
            - lack of neuromuscular co-ordination or strength
            - cognitive impairment
            - sedative medication and alcohol
        - secondary causes of osteoporosis such as:
            - rheumatoid arthritis and other inflammatory arthropathies (risk independent of BMD and steroid use)
            - prolonged immobilisation or very sedentary lifestyle
            - primary hypogonadism (men and women)
            - primary hyperparathyroidism
            - hyperthyroidism
            - post-transplantation
            - chronic kidney disease
            - gastrointestinal disease e.g. Crohn's, ulcerative colitis, coeliac disease
            - untreated premature menopause (<45) or prolonged secondary amenorrhoea
            - type 1 and type 2 diabetes
            - chronic liver disease

                - COPD
- presentation
    - most common sites for fragility fractures are:
        - vertebrae
        - hip (proximal femur)
        - wrist (distal radius)
    - other sites include:
        - pelvis
        - ribs
        - arms
        - shoulder
    - vertebral compression fractures may not be noticed at the time
- consequences
    - mobility and independence may be affected
    - potential for loss of confidence, anxiety, depression, reduced quality of life
    - compression fractures can cause:
        - pain
        - morbidity associated with high doses of analgesia
        - loss of height
        - difficulty breathing
        - loss of mobility
        - gastrointestinal symptoms
        - difficulty sleeping
        - symptoms of depression
- underdiagnosis of vertebral fractures
    - National Osteoporosis Society guidelines highlighted reasons for underdiagnosis:
        - only a minority result from a fall
        - symptoms are often attributed to another cause by both patient and healthcare professional
        - the need for spinal imaging for a patient with risk factors for osteoporosis presenting with new back pain is often not recognised
        - if imaging is undertaken for other reasons the spine may not be scrutinised in the reporting process
        - vertebral fractures may be reported using confusing and ambiguous terminology
        - the clinician may regard the fracture as incidental to the presenting complaint and fail to recognise the clinical importance
- management
    - multidisciplinary care is required
        - both orthopaedic and medical care may be required
    - management of the acute injury
        - pain relief
        - management of associated chronic disease
        - fluid management
        - fracture stabilisation and/or surgery as indicated
            - comorbidity and pre-fracture condition should be taken into account when surgical decisions are made

- pre-operative investigations would include:
    - CXR
    - ECG
    - FBC, blood group, clotting studies
    - renal function
    - glucose
    - assessment of cognitive function
- most vertebral fractures are managed in the community with analgesia and physiotherapy and do not require admission
    - vertebroplasty and kyphoplasty are surgical options where conservative measures fail

- postoperative care includes:
    - pain control
    - antibiotic prophylaxis where appropriate
    - monitoring of FBC and correction of anaemia where appropriate
    - routine systems examination to detect early complications or exacerbation of chronic conditions
    - regular assessment of cognitive function
    - prevention and management of pressure sores
    - monitoring of nutritional status and renal function
    - monitoring of bladder and bowel function and management of problems as required
    - wound care
    - early mobilisation
- rehabilitation and education
    - early physiotherapy and muscle strengthening exercises
    - fall prevention measures
    - balance training
    - medication review
    - education about modifiable risk factors (smoking, weight, alcohol, diet, exercise etc)
    - education about treatment, duration of treatment and follow up
- assessment of risk for future fragility fracture
    - would involve assessing risk factors and possibly DEXA scanning
        - NICE guidelines suggest that in a patient over 75 with a fragility fracture a diagnosis of osteoporosis can be assumed without DEXA scanning and treatment started
        - SIGN guidelines advise DEXA scan following fragility fracture and treat if osteoporosis confirmed
- treatment of low bone density (from hospital or GP)
    - supplement calcium and vitamin D if required
        - no risk has been identified of calcium supplementation when combined with vitamin D, and the combination is more effective than vitamin D alone in preventing fractures
    - where high risk of future fragility fracture is identified by a calculator combining BMD with clinical risk factors, treatment at improving BMD may be started, e.g.:

- bisphosphonates
    - alendronate or risedronate orally daily or weekly
    - ibandronate orally monthly or IV 3 monthly
    - zoledronic acid by IV infusion annually
- denosumab
    - monoclonal antibody which reduces osteoclast activity
    - approved by NICE for secondary prevention for postmenopausal women with increased risk of fractures who cannot comply with special instruction for alendronate/risedronate or have an intolerance or contraindication
- strontium ranelate
    - only used for severe osteoporosis where other treatment options are not possible due to increased risk of myocardial infarction
    - cannot be used if there are cardiovascular risk factors
- raloxifene
    - selective oestrogen receptor modulator
    - inhibits bone resorption
    - used for prevention and treatment of osteoporosis in post menopausal women
    - reduces risk of vertebral but not other fractures
- teriparatide
    - recombinant fragment of parathyroid hormone
    - sometimes prescribed by secondary care for patients at very high risk who cannot have bisphosphonates
- hormone replacement therapy
    - option in younger perimenopausal women who also need treatment for symptoms of menopause

- guidelines for assessment and management differ between organisations and evidence of benefit for reduction of fractures in over 80s is not there

EIP5 frailty

Clinical Frailty Score (Rockwood)

- very fit
    - robust, active, energetic and motivated
    - commonly exercise regularly
    - among the fittest for their age
- well
    - no active disease symptoms but less fit than category 1
    - often they are seasonally active or exercise occasionally
- managing well
    - medical problems are well controlled
    - not regularly active beyond routine walking
- vulnerable
    - not dependent on others for daily help but symptoms often limit activities
    - common complaint is being ‘slowed up’ and/or being tired during the day

- mildly frail
    - often have more evident slowing and need help in higher order IADLs (finances, transportation, heavy housework, medications)
    - typically, mild frailty progressively impairs shopping and walking outside alone, meal preparation and housework
- moderately frail
    - need help with all outside activities and with keeping house
    - often have problems with stairs and need help with bathing
    - might need minimal assistance (cuing, standby) with dressing
- severely frail
    - completely dependent for personal care from whatever cause (physical or cognitive)
    - seem stable and not high risk of dying within around 6 months
- very severely frail
    - completely dependent, approaching the end of life
    - typically could not recover even from a minor illness
- terminally ill
    - approaching the end of life
    - all people with a life expectancy <6 months who are not otherwise evidently frail
- only validated in older patients (65 and over)
- collateral history can be very valuable
- if acutely unwell, they should be scored on how they were two weeks ago, not today
- history is crucial

EIP6 hypothermia

- background
    - factors leading to altered thermoregulation in the older adult
        - cold exposure due to:
            - poverty
            - climate
            - inadequate clothing
            - inadequate heating systems
        - lack of fans or air conditioning in hot conditions
        - reduced heat production from lower metabolic rate
        - less subcutaneous fat and muscle mass resulting in reduced insulation
            - reduced shivering due to lower muscle mass
        - alcohol, sedative drugs and cognitive impairment alter temperature perception, thirst and reduce shivering
        - poor mobility or falls may lead to prolonged exposure to cold or hot conditions
        - reduced number and function of sweat glands limiting ability to reduce core temperature
        - reduced autonomic nervous system activity resulting in reduced shivering, sweating and vasoconstriction or vasodilatation
        - antihypertensives and other medication may limit vasoactive response to temperature

        - infection may precipitate or complicate hypothermia or hyperthermia
    - hypothermia may follow a fall or period of immobility in a cold environment
    - defined as an involuntary drop in core body temperature to <35°
        - mild (<35°)
        - moderate (<32°)
        - severe (<30°)
    - primary hypothermia
        - inadequate heat conservation due to excessive cold
    - secondary hypothermia
        - due to another disease process such as:
            - sepsis
            - hypothyroidism
            - adrenal insufficiency
- initial assessment
    - A+B
        - there may be reduced consciousness and respiratory depression
        - attach high flow oxygen
        - consider airway interventions/ventilatory support
    - C
        - eventually results in myocardial suppression, bradycardia and hypotension
        - AF with reduced ventricular rate is common
        - VF and asystolic arrest may occur
        - continuous cardiac monitoring is required
    - D
        - there may be neurological signs such as confusion, ataxia, seizures, focal neurological deficit, speech disturbance
    - complications include:
        - impaired coagulation
        - increased risk of pancreatitis
        - renal failure
        - multi-organ failure
    - assess for contributing or consequential medical conditions such as pneumonia or other infections
    - assess for any injuries that may have contributed or caused the problem
- investigations
    - bloods
        - FBC/U&E/LFT/coag
        - CK
        - TFTs
        - blood glucose
        - amylase
        - VBG/ABG
    - blood and urine cultures if sepsis suspected
    - ECG
        - may show prolonged PR, J waves, AF
- management
    - treat precipitating causes (e.g. broad spectrum antibiotics)

    - gradual rewarming at 0.5-1°/hour
        - rapid rewarming may lead to hypotension, cerebral and pulmonary oedema or arrhythmias
        - there may be afterdrop – a further drop in core temperature as cool blood returns from the peripheries
    - passive rewarming
        - with mild hypothermia, stable circulation and shivering: dry clothes and warm blankets
    - active external rewarming
        - hot air blankets to reduce conductive heat losses and provide a heat source – indicated for mild to moderate hypothermia
    - active internal rewarming
        - warm IV fluids (43°) and warm humidified oxygen
        - in severe hypothermia consider bladder, peritoneal or pleural cavity irrigation with warm fluid
        - ECMO and cardiopulmonary bypass can be considered
- cardiac arrest
    - adjustments for hypothermia:
        - look for signs of life and at the ECG trace for up to 1 min before determining there is no cardiac output and starting CPR
        - do not give adrenaline or other arrest drugs until the core temperature is over 30°
            - double the time interval between drug doses until 35°, then resume the usual protocol
        - in shockable rhythms, give a maximum of 3 shocks if the temperature remains below 30° - further attempts should be delayed until the temperature arises above this threshold

ElP7 incontinence

- urinary incontinence
    - definitions
        - functional incontinence
            - patient unable to reach the toilet in time
        - stress incontinence
            - involuntary leakage of urine on effort or exertion, sneezing or coughing
            - due to an incompetent sphincter
            - may be associated with genitourinary prolapse
        - urge incontinence
            - involuntary urine leakage accompanied by or immediately preceded by urgency of micturition
                - a sudden and compelling desire to urinate that cannot be deferred
            - detrusor instability or hyperreflexia leads to involuntary detrusor contraction
            - may be idiopathic or secondary to neurological processes such as stroke, Parkinson's disease, MS, dementia, spinal cord injury

            - sometimes caused by local irritation due to infection or bladder stones
        - mixed incontinence
            - involuntary leakage of urine associated with both urgency and exertion, effort, sneezing or coughing
        - overactive bladder syndrome
            - urgency that occurs with or without urge incontinence and usually with frequency and nocturia
            - may be 'wet' or 'dry' depending on whether it is associated with incontinence
            - usually caused by detrusor overactivity
        - overflow incontinence
            - usually due to chronic bladder outflow obstruction
            - often due to prostatic disease in men
            - requires early assessment and intervention due to risk of obstructive nephropathy caused by back pressure
            - may also be due to a neurogenic bladder
        - true incontinence
            - may be due to a fistulous track between the vagina and the ureter, bladder or urethra
            - there is continuous leakage of urine
    - risk factors
        - women
            - pregnancy and vaginal delivery
            - diabetes
            - oral oestrogen therapy
            - high BMI
            - hysterectomy
            - recurrent UTIs
        - men
            - lower urinary tract symptoms
            - infections
            - functional and cognitive impairment
            - neurological disorders
            - prostatectomy
        - both sexes
            - neurological disease
            - cognitive impairment
            - obstruction (e.g. large prostate or pelvic mass)
            - stool impaction
    - history
        - leakage of urine on sneezing, coughing, exercise, rising from sitting, lifting
        - urgency and failure to reach a toilet in time
        - frequency of urine during the day/night
        - dribbling of urine after leaving the toilet
        - loss of bladder control
        - feeling of incomplete bladder emptying

        - dysuria
        - bladder spasms
        - factors likely to affect management, e.g. mobility, hand co-ordination, cognitive function, social support, lifestyle
        - obstetric history
        - advice to complete a bladder chart for at least 3 days (including working days and days off)
        - sexual dysfunction and quality of life
        - functional status and access to a toilet
        - whether any medication contributes to symptoms
        - bowel habit
        - desire for treatment
    - examination
        - women
            - digital assessment of pelvic floor muscle contraction
            - bimanual/vaginal examination to assess for presence of prolapse
            - signs of vaginal atrophy
            - abdominal, pelvic and neurological examination
        - men
            - digital rectal examination to assess prostate shape, size and consistency and check for other rectal pathology
            - digital anal assessment can give an indication of pelvic floor muscle strength in men
            - abdominal, pelvic and neurological examination
    - possible investigation
        - urine dip
        - MSU for culture and sensitivities if symptomatic of UTI with positive dip (or symptoms and negative dip)
        - send MSU if no symptoms but dip positive but do not start treatment until results available
        - consider U&Es
        - post void residual volume
        - specialist investigations may include urodynamics and ultrasound KUB if there is a neurological disease where renal complications could occur; two week wait referral if there is incontinence alongside symptoms suggestive of bladder or renal cancer
    - management
        - pads or collecting devices to achieve social continence
            - may be required permanently if other methods of management have been excludes
        - overactive bladder
            - reduction in caffeine intake
            - modification in high or low fluid intake
            - weight loss if BMI >30
            - bladder training
                - first line treatment for at least 6 weeks

                        - pelvic muscle training, scheduled voiding intervals with stepped increases, suppression of urge with distraction or relaxation techniques
                - drug treatment
                    - anticholinergics such as oxybutynin only in younger patients
                    - intravaginal oestrogens in post-menopausal women with vaginal atrophy
                    - mirabegron to promote detrusor relaxation
            - stress incontinence
                - pelvic floor muscle exercises
                    - a three month trial of 8 contractions 3 times a day
                    - electrical stimulation and/or biofeedback can be considered
                - drug treatment
                    - duloxetine as a second line treatment
                - surgical treatment
                    - may be considered if other measures fail
            - mixed incontinence
                - management to target the main symptom
                    - pelvic floor exercises and bladder training
                - antimuscarinics such as oxybutynin should be avoided in the elderly as they can adversely affect cognitive performance
            - overflow incontinence
                - relief or treatment of the obstruction
                - may require intermittent self-catheterisation
- faecal incontinence
    - risk factors
        - patients with diarrhoea
        - patients with anal problems
            - third and fourth degree obstetric injury
            - rectal or pelvic organ prolapse
            - pelvic radiotherapy or colonic resection
            - perianal itching/soreness/pain or anal surgery
        - patients with urinary incontinence
        - frail elderly patients
        - patients with neurological problems or spinal disease
        - patients with severe cognitive impairment or learning difficulties
    - management
        - condition-specific
            - treat potentially reversible causes where identified:
                - faecal loading
                - potentially treatable causes of diarrhoea, e.g. infection, IBD, irritable bowel syndrome
                - warning signs for lower GI cancer
                - rectal prolapse or third-degree haemorrhoids
                - acute anal sphincter injury
                - acute disc prolapse/cauda equina syndrome
        - basic initial interventions

- diet
    - food and fluid diary with modification of one food at a time
    - at least 1.5 litres of fluid/day (unless contraindicated) if there are hard stools or dehydration
    - malnutrition screening
- bowel and toileting habits
    - encourage bowel emptying after a meal
    - private and comfortable toilet facilities
    - sitting or squatting position to avoid straining
    - easily removable clothing and assistance with mobilising to toilet
- medication
    - antidiarrhoeals if other causes have been excluded
        - loperamide first line, titrated until desired stool consistency reached
        - codeine as an alternative
- coping strategies
    - advice on:
        - continence products
        - emotional and psychological support
        - talking to friends and family
        - planning travel and carrying a RADAR key
        - disposable pads
        - anal plugs
        - skin care, odour control, laundry advice
        - disposable gloves
- specialist management
    - may include:
        - pelvic floor muscle training
        - bowel retraining
        - dietary assessment and management
        - biofeedback
        - electrical stimulation
        - rectal irrigation
        - anal bulking agents
        - surgery (e.g. sphincter repair)

ElP8 increasing care needs

- elements of systematic enquiry
    - falls
    - pain
    - incontinence
    - memory loss
    - depression
    - anxiety
    - eyesight and hearing change
    - weight loss

    - change in eating pattern
    - sleep disturbance
- quick functional screen
    - anyone coming in to help at home
        - ask directly about professional carers, district nurses, relatives, neighbours
        - how often they come and how many times a day
    - any walking aids
        - any wheelchair use
        - what happens for long distances
    - any help in washing or dressing
    - any help using the toilet and whether they wear pads
    - any help with shopping/cooking
    - who does the housework
    - how they manage their tablets
    - who sorts their bills out
    - whether they get our and about
        - day centres
        - driving
- components of comprehensive geriatric assessment
    - medical
        - active medical problems
        - comorbid conditions and disease severity
        - medication review
        - nutritional status
    - mental health
        - cognitive assessment
        - mood and anxiety
    - functional capacity
        - activities of daily living and instrumental activities of daily living
            - basic
                - bathing
                - dressing
                - toileting
                - transfers
                - continence
                - feeding
            - instrumental
                - shopping
                - cooking
                - housework
                - telephone use
                - transport
                - medication
                - finances
        - activity levels and exercise tolerance
        - gait and balance
    - social circumstances
        - social support from family, neighbours or friends

        - daytime activities and social network
        - eligibility for care resources
    - environment
        - home situation, facilities and safety
        - use of local resources and community services
        - transport facilities
- community care services
    - background
        - enable people to remain in their own homes with as much independence as possible
        - provided or arranged by local authority social services
        - provision involves matching client expectation, available finances and people willing to do the job
            - finance rules are complicated and the Citizens Advice Bureau can be helpful
        - available services may include:
            - home care services
                - help with personal tasks
                    - bathing
                    - washing
                    - getting up and going to bed
                    - shopping
                    - managing finances
            - home helps
                - assistance with general domestic tasks including cleaning and cooking
                - may be important in maintaining hygiene in the home
            - adaptations to the home
                - major adaptations
                    - e.g. installation of stairlift or downstairs lavatory, lowering worktops in the kitchen
                - minor adaptations
                    - e.g. handrails in the bathroom
            - meals
                - deliveries of meals or provision at day centres or lunch clubs
            - recreational, occupational, educational and cultural activities
    - National Health Service and Community Care Act 1990
        - devolved the responsibility for means-tested funding to local social services departments
        - any person can make a referral to Social Services on behalf of the patient
        - the local authority carries out an assessment and provides a written care plan with:
            - the services to be provided, by whom and when and what will be achieved by providing them
            - a contact point to deal with problems
            - information on how the person can request a review of the services if circumstances change

        - the Community Care (Residential Accommodation) Act 1998 restricts the amount of a person's capital which may be taken into account in determining whether the patient should be provided with residential accommodation

EIP9 memory loss

- the 4AT assessment
    - alertness
        - observe the patient and, if asleep, awake with speech or gentle touch on the shoulder
        - ask them to state their name and address
        - scoring
            - normal (fully alert but not agitated throughout assessment): 0
            - mild sleepiness for <10 sec after waking, then normal: 0
            - clearly abnormal: 4
    - AMT4
        - age, date of birth, place (name of hospital or building), current year
            - no mistakes: 0
            - one mistake: 1
            - two or more mistakes/untestable: 2
    - attention
        - ask the patient to state the months of the year in backwards order starting from December
        - to assist initial understanding, one prompt of 'what is the month before December?' is permitted
        - scoring
            - 7 months or more correctly: 0
            - starts but scores <7 months/refuses to start: 1
            - untestable (cannot start because unwell, drowsy, inattentive): 2
    - acute change or fluctuating course
        - evidence of significant change or fluctuation in alertness, cognition, other mental function (e.g. paranoia, hallucinations) arising over the last 2 weeks and still evident in last 24h
        - scoring
            - no: 0
            - yes: 4
    - interpretation
        - 4 or above: possible delirium +/- cognitive impairment
        - 1-3: possible cognitive impairment
        - 0: delirium or severe cognitive impairment unlikely (but delirium still possible if information about acute change/fluctuating course incomplete
- mild cognitive impairment
    - criteria for diagnosis
        - concern regarding a change in cognition form the patient, a knowledgeable informant or from a skilled clinician observing the patient

        - objective evidence of impairment (from cognitive testing) in one or more cognitive domains, including memory, executive function, attention, language or visuospatial skills
        - preservation of independence in functional abilities (although individuals may be less efficient and make more errors at performing ADLs)
        - no evidence of a significant impairment in social or occupational functioning (i.e. not dementia)
    - characteristics suggesting that MCI is due to Alzheimer's disease
        - memory impairment present
        - progressive decline in cognition over months to years
            - very rapid decline may suggest prion disease, neoplasm or metabolic disorders
        - lack of Parkinsonism and visual hallucinations (suggestive of dementia with Lewi bodies)
        - lack of vascular risk factors and extensive cerebrovascular disease on brain imaging (suggestive of vascular cognitive impairment)
        - lack of prominent behavioural or language disorders (suggestive of frontotemporal lobar degeneration)
    - assessment of cognitive impairment
        - screening tools include:
            - Mini Mental State Examination
            - Six-item Cognitive Impairment Test (6CIT)
            - Abbreviated Mental Test (AMT)
    - aetiology
        - the most common cause of significant cognitive impairment is Alzheimer's disease, which may begin with mild impairment
        - factors influencing cognitive decline may include:
            - cerebrovascular events
            - hypothyroidism
            - hyperparathyroidism/hypoparathyroidism
            - hypoperfusion (e.g. heart failure)
            - head trauma (including recent trauma or previous boxing)
            - folate, B12 and B6 deficiency
            - open heart surgery with cardiopulmonary bypass
            - medication use, particularly sedatives
            - hepatic impairment
            - sleep disorders, e.g. obstructive sleep apnoea
            - depression
            - psychological stress
            - drug or alcohol abuse
            - toxins, infections, metabolic and structural causes
            - oestrogen decline
            - high corticosteroid levels
    - investigations
        - to rule out physical causes
            - full history
                - past medical history

        - family history
        - drug history
        - social history
    - full examination
        - consideration of cardiac or neurological abnormalities
    - assessment of cognition
    - laboratory tests
        - FBC
        - U&Es
        - LFTs
        - calcium
        - B12
        - TFTs
        - random or fasting blood sugar
  - specialist investigations may include
    - neuropsychological testing
    - EEG and evoked potentials
    - imaging
        - functional imaging
        - CT
        - PET scan
        - MRI
- management
  - there is no medication to manage mild cognitive impairment
  - coping with everyday life
    - 'to do' lists of tasks
    - breaking down tasks into manageable chunks
    - trying to do one thing at a time to reduce confusion
    - having a routine to give structure to the day
    - taking time and not hurrying
  - memory aids
    - clocks, watches, daily newspapers
    - keeping a diary of appointments and lists
    - using sticky-backed notes
    - keeping important things such as keys and money in the same place to make them easier to find
    - keeping important telephone numbers by the phone
    - arranging to pay regular bills by direct debit or standing order
    - trying not to be embarrassed about forgetting things
    - avoiding trying too hard to remember something specific
    - talking to friends and family about how they can help
    - assistance from an occupational therapist
  - general health
    - regular exercise
    - smoking cessation
    - adequate but not excessive sleep
- referral

        - may be appropriate from primary care when it is suspected that the cognitive impairment is more than just minor and/or concerns about dementia which may require specialist intervention
        - NICE recommendations include:
            - individual's self-report of changes in memory, capability or mood
            - informant history that support self-report and add new significant details
            - exclusion of depression and delirium as primary pathologies
            - measurable cognitive losses using a standardised instrument
            - absence of red flag symptoms suggesting alternative diagnosis (e.g. urinary incontinence/ataxia alongside cognitive impairment)
    - prognosis
        - mild cognitive impairment has a risk of developing dementia of 10-15% per year

ElP10 unsteadiness/balance disturbance

- vertigo
    - background
        - perception of movement where no movement exists
        - mismatch or asymmetric activity of visual, vestibular and/or proprioceptive systems
        - peripheral causes must be distinguished from central
            - peripheral: CN VIII, vestibular apparatus
            - central: brainstem, cerebellum
    - classification
        - triggered episodic vestibular syndrome
            - triggered by movement
                - change in body position, head movement, valsalva
            - lasts seconds to hours with asymptomatic periods in between
            - benign:
                - BPPV
                - orthostatic hypotension
            - dangerous:
                - posterior fossa tumour
        - spontaneous episodic vestibular syndrome
            - distinct onset
            - lasts minutes to hours
            - typically asymptomatic on presentation
            - benign:
                - anxiety
                - vasovagal syncope
                - Ménière's disease
            - dangerous:
                - TIA
                - arrhythmia
                - PE
        - acute vestibular syndrome

        - abrupt and persistent
        - can be exacerbated by movement but not triggered by it
            - symptoms persist at rest
        - benign:
            - vestibular neuritis
            - labyrinthitis
        - dangerous:
            - posterior stroke
        - use HINTS exam to differentiate if patient symptomatic
- differential
    - vestibular/otologic
        - benign paroxysmal positional vertigo (BPPV)
        - traumatic
            - following head injury
        - infection
            - labyrinthitis
            - vestibular neuritis
            - Ramsay Hunt syndrome
        - Ménière's disease
        - ear foreign body
        - otic barotrauma
        - otosclerosis
    - neurologic
        - cerebellar stroke
        - vertebrobasilar insufficiency
        - anterior inferior cerebellar artery syndrome
        - neoplasm: cerebellopontine angle tumours
        - basal ganglion diseases
        - vertebral artery dissection
        - multiple sclerosis
        - infections
            - neurosyphilis
            - tuberculosis
        - epilepsy
        - basilar migraine
    - other
        - haematologic
            - anaemia
            - polycythaemia
            - hyperviscosity syndrome
        - toxic
            - alcohol
            - aminoglycosides
        - chronic renal failure
        - metabolic
            - thyroid disease
            - hypoglycaemia
- evaluation
    - glucose
    - neurological examination

        - ear examination
        - consider CTA or MRA
        - HINTS exam
            - only in a patient with acute persistent vertigo, nystagmus and a normal neurological exam
            - head impulse test
                - reassuring if abnormal (corrective saccade)
            - nystagmus
                - reassuring if unidirectional, horizontal
            - test of skew
                - reassuring if no skew deviation
        - Unterberger's test
            - used to identify damage to one of the labyrinths
            - patient marches on the spot for 30 seconds with eyes closed – observe for lateral rotation
            - if there is no rotation there is symmetrical labyrinth function
            - if there is labyrinthine damage, the patient rotates to the side of the damage
        - Romberg's test
            - used to identify instability of central or peripheral cause
            - patient stands up straight with feet together and arms outstretched, then shuts their eyes
            - if they are unable to maintain balance with eyes closed, the test is positive
                - they usually fall to the side of the lesion – be prepared to support them
                - a positive test suggests a problem with proprioception or vestibular function
                    - can also be positive in neuromuscular disorders and may not be reliable in very elderly people
    - management
        - peripheral
            - symptomatic control
                - antihistamines
                    - inhibit vestibular stimulation and vestibular-cerebellar pathways
                    - diphenhydramine (Benadryl)
                - anticholinergics
                    - scopolamine transdermal patch behind ear
                - antidopaminergics
                    - metoclopramide
                - benzodiazepines
                    - diazepam PRN QDS
            - cause reversal
                - Epley manoeuvre for BPPV
        - central
            - rule out CVA
            - MRI
            - rule out vascular insufficiency
- dizziness
    - causes

- cardiovascular
    - postural hypotension
    - cerebrovascular disease
    - carotid sinus syndrome
    - vertebrobasilar insufficiency
    - aortic stenosis
    - subclavian steal syndrome
    - cardiac arrhythmias
- neurological
    - following head injury
    - epilepsy
    - multiple sclerosis
    - Parkinsonism
    - dementia
    - brain tumours
        - especially brainstem and cerebellar
    - benign intracranial hypertension
    - normal pressure hydrocephalus
    - peripheral neuropathy
- otological
    - Ménière's disease
    - BPPV
    - vestibular neuritis and labyrinthitis
    - vestibular migraine
        - usually has attacks of spontaneous or positional vertigo lasting seconds to days with associated migraine symptoms
    - otosclerosis and Paget's disease of bone
    - middle ear trauma
    - following surgery
        - e.g. stapedectomy, cochlear implant
    - tumours, cholesteatoma
- metabolic
    - hypoglycaemia
    - adrenal insufficiency
    - hypothyroidism
- haematological
    - anaemia
    - hyperviscosity
- psychogenic
    - generalised anxiety
    - agoraphobia
    - panic attacks
    - hyperventilation
- miscellaneous
    - viral illness
    - migraine headaches
    - other infections, e.g. acute bacterial infections, Lyme disease, HIV
    - ocular: visual impairment
    - cervical, e.g. cervical spondylosis
    - multisensory dizziness syndrome

                - occurs when there are reduced inputs from more than one sensory system
                - e.g. reduced vision, vestibular dysfunction, peripheral neuropathy, autonomic neuropathy
            - autoimmune/connective tissue disorders, e.g. rheumatoid arthritis, SLE
            - drug intoxication
                - e.g. acute drug or alcohol intoxication, carbon monoxide poisoning, chronic alcohol misuse
            - iatrogenic – side effects of medication, e.g. antihypertensives, antidepressants, aminoglycosides, antiarrhythmics
    - categorisation
        - vertigo
            - abnormal sensation of movement, either of the surroundings or the person
        - presyncope
            - feeling of lightheadedness, muscular weakness and feeling faint
        - disequilibrium
            - sensation of unsteadiness, not localised to the head, that occurs with walking and is relieved by rest
            - most common cause is multiple sensory deficits in elderly patients who may have deficits in all three balance-preserving senses (vestibular, visual and proprioceptive)
        - nonspecific dizziness
            - vague complaint of dizziness with no features pointing to one of the other categories
    - management
        - may include:
            - vestibular rehabilitation
            - psychological intervention such as CBT
            - medication for symptomatic control
            - occasionally surgery (e.g. for intracranial abscess, acoustic neuroma, perilymph fistula)

EIC1 comprehensive geriatric assessment

- definition
    - 'a multidimensional, interdisciplinary diagnostic process to determine the medical, psychological and functional capabilities of a frail older person in order to develop a coordinated and integrated plan for treatment and long term follow up'
    - often uses standardised assessment tools
    - leads to:
        - improved discharge rates
        - reduced readmissions
        - reduced long-term care
        - greater patient satisfaction
        - lower costs
- components
    - medical
        - active medical problems

        - comorbid conditions and disease severity
        - medication review
        - nutritional status
    - mental health
        - cognitive assessment
        - mood and anxiety
    - functional capacity
        - activities of daily living and instrumental activities of daily living
            - basic
                - bathing
                - dressing
                - toileting
                - transfers
                - continence
                - feeding
            - instrumental
                - shopping
                - cooking
                - housework
                - telephone use
                - transport
                - medication
                - finances
        - activity levels and exercise tolerance
        - gait and balance
    - social circumstances
        - social support from family, neighbours or friends
        - daytime activities and social network
        - eligibility for care resources
    - environment
        - home situation, facilities and safety
        - use of local resources and community services
        - transport facilities
- age-associated physiological changes
    - changes in body composition
        - reduction in muscle bulk and lean body mass (sarcopenia)
        - body fat may increase
    - reduction in bone mass and strength with increased risk of fracture; osteoarthritic changes in joints
    - reduction in blood volume; reduced tolerance of tachycardia; reduced ability to control blood pressure with postural changes
    - reduction in ventilatory capacity
    - reduction in kidney function; impaired thirst mechanisms increasing susceptibility to dehydration
    - reduced sensitivity to vitamin D and subsequent reduction in calcium absorption
    - reduced motility of the large bowel; reduced hepatic mass and blood flow (may affect hepatic metabolism of drugs)

    - nervous system changes including reduction in cortical function and reduced motor and sensory peripheral nerve function; changes in autonomic function, including control of heart rate and temperature regulation
    - reduced elasticity of the eye's lens; high tone hearing impairment
- validated tools for assessment of disability or needs in elderly people include:
    - Barthel's Index
    - Northwick Park Dependency Scale
    - Camberwell Assessment of Need in the Elderly
- aspects of management
    - treat contributing causes
        - uncontrolled cardiac, respiratory or metabolic disease
        - reversible causes of hearing loss (e.g. wax)
        - potentially treatable neurological disease (e.g. tumours)
    - drug treatment
        - medication can contribute to the problem and the solution
        - polypharmacy
        - vitamin D deficiency
    - surgical treatment
        - may include joint replacement, cataract surgery, surgery for prostatic hypertrophy
    - provision of aids and appliances
        - home adjustments such as grip rails, stair lifts, removal of dangers such as loose carpets
        - vision and hearing aids
        - adapted safety devices
    - pain management
    - social and environmental interventions
        - financial support
            - e.g. access to benefits and grants
        - social support
            - e.g. day centres, social activities and befriending
        - housing support

EIC2 acute confusion

- background
    - delirium is difficult to define but involves abnormalities or thought, perception and level of awareness
    - up to a third of cases can be avoided
    - important points to remember include:
        - patients are vulnerable
        - the diagnosis is easy to miss and can rapidly worsen
        - if collateral history not available, treat as acute confusion until proved otherwise
        - full physical examination and observations are required
        - check blood glucose
- risk factors
    - age ≥65

    - male
    - pre-existing cognitive deficit
    - severity of dementia
    - severe comorbidity
    - previous delirium
    - operative factors (e.g. hip fracture repair, emergency surgery)
    - certain conditions (e.g. burns, AIDS, fractures, infection, low albumin, dehydration)
    - drug/alcohol use and dependence
    - hypo- or hyperthermia
    - visual or hearing problems
    - poor mobility
    - social isolation
    - stress
    - terminal illness
    - movement to new environment
    - ICU admission
- causes
    - acute infection
        - UTI
        - pneumonia
        - sepsis
        - viral infection
        - meningitis
        - encephalitis
        - cerebral abscess
        - malaria
    - prescribed drugs
        - benzodiazepines
        - analgesics (e.g. morphine)
        - anticholinergics
        - anticonvulsants
        - anti-parkinsonian medications
        - steroids
    - surgical
        - post-operative
    - toxic substances
        - substance misuse or withdrawal
        - carbon monoxide poisoning
        - exposure to heavy metals
        - barbiturate withdrawal
    - vascular disorders
        - cerebrovascular haemorrhage or infarction
        - cardiac failure or ischaemia
        - subdural haemorrhage
        - subarachnoid haemorrhage
        - vasculitis (e.g. SLE)
        - cerebral venous thrombosis
        - migraine

    - metabolic
        - hypoxia
        - electrolyte abnormality
        - hypo- or hyperglycaemia
        - hepatic impairment
        - renal impairment
    - vitamin deficiency
        - thiamine
        - nicotinic acid
        - B12
    - endocrinopathies
        - hypo- or hyperthyroidism
        - hypopituitarism
        - hypo- or hyperparathyroidism
        - Cushing's disease
        - porphyria
        - carcinoid
    - trauma
        - head injury
    - epilepsy
        - e.g. post-ictal
    - neoplasia
        - brain tumour
        - brain metastases
        - paraneoplastic syndromes
    - other
        - urinary retention
        - faecal impaction
        - multiple aetiology
        - unknown aetiology
- features
    - usually acute or subacute presentation
    - fluctuating course
    - clouded consciousness/impaired cognition/disorientation
    - poor concentration
    - memory deficit – mainly short-term memory
    - abnormalities of sleep-wake cycle
    - abnormalities of perception (e.g. hallucinations, illusions)
    - agitation
    - emotional lability
    - psychotic ideas – short duration and simple content
    - neurological signs – e.g. unsteady gait, tremor
- subtypes
    - hypoactive
        - apathy and quiet confusion
        - easily missed
        - can be confused with depression
    - hyperactive

        - agitation
        - delusions
        - disorientation
        - can be confused with schizophrenia
    - mixed
- CAM (Confusion Assessment Method) criteria for delirium
    - confusion that has developed suddenly and fluctuates AND
    - inattention (ask if they are easily distracted or have problems focusing attention) AND EITHER
    - disorganised thinking OR
    - altered level of consciousness
- differential
    - dementia
    - depression
    - bipolar disorder
    - functional psychoses e.g. schizophrenia
    - thyroid disease
    - non-convulsive epilepsy
    - Charles Bonnet syndrome
- investigations
    - may include:
        - bloods
            - FBC
            - U&Es
            - glucose
            - calcium
            - magnesium
            - LFTs
            - TFTs
            - cardiac enzymes
            - B12 levels
            - syphilis serology
            - autoantibody screen
        - urine dipstick and microbiology if symptomatic
        - blood cultures if indicated
        - ECG
        - pulse oximetry +/- ABG
        - CXR if indicated
        - CT head if indicated
        - lumbar puncture if indicated
- treatment and management
    - manage in the community if possible as moving to hospital likely to worsen symptoms
    - supportive management
        - clear communication
        - reminders of day, time, location and identity of people
        - clock available

        - familiar objects from home
        - glasses, hearing aids and walking aids
        - staff consistency
        - relaxation such as watching television
        - involve family and carers
    - environmental measures
        - avoid over- and under-stimulation
        - adequate space and sleep
            - try to maintain normal sleep-wake cycle
            - avoid hypnotics
        - single room if possible
        - avoid jargon
        - control excess noise
        - control room lighting with low wattage bulb at night
        - control room temperature (aim for 21-23 degrees)
        - use health advocates/interpreters as needed
        - maintain competence (e.g. encourage ambulation)
        - adequate nutrition
        - use least restrictive management for wandering
            - try to identify cause – e.g. need for toilet
            - use distraction/family
    - medical management
        - correct underlying problems including:
            - infection
                - however it is often overdiagnosed, especially UTI
            - constipation
            - urinary retention
                - often missed as patients may still pass urine
                - address constipation, anticholinergic use and immobility
                - if catheter required, plan to remove as early as possible
            - dehydration and electrolyte abnormalities
                - encourage oral rehydration if possible
            - pain
                - regular paracetamol
                - consider a weak opioid but monitor for worsening of delirium
            - medication
                - review and optimisation
    - pharmacological management
        - should be avoided if possible
        - antipsychotics may be helpful for patients not responding to verbal and non-verbal de-escalation techniques
            - haloperidol and olanzapine are preferred
            - lowest dose for shortest period of time (usually a week or less)
            - both can cause extra-pyramidal side effects and should be avoided in patients with Parkinson's or Lewy-Body dementia
        - benzodiazepines for alcohol withdrawal

        - diazepam or chlordiazepoxide
- common drug causes of delirium
    - benzodiazepines
    - narcotics
    - first-generation antihistamines
    - antispasmodics
    - fluoroquinolones
    - warfarin
    - captopril
    - theophylline
    - isosorbide dinitrate
    - dipyridamole
    - furosemide
    - lithium
    - tricyclic antidepressants
    - cimetidine
    - anti-arrhythmics
    - statins
    - digoxin
    - steroids
    - beta-blockers
    - over the counter medications such as those containing alcohol or chlorphenamine
- complications
    - hospital-acquired infection
    - pressure sores
    - fractures from falls
    - residual cognitive impairment
    - some progress to stupor, coma and death

EIC3 ceiling of care and end of life care

NICE end of life care guideline

- quality statements
    - adults who are likely to be approaching the end of their life are identified using a systematic approach
    - adults who are approaching the end of their life have opportunities to discuss advance care planning
    - adults approaching the end of their life receive care that is coordinated between health and social care practitioners within and across different services and organisations
    - adults approaching the end of their life and their carers have access to support 24 hours a day 7 days a week
    - carers providing end of life care to people at home are supported to access local services that can provide assistance
- definition of adults approaching the end of life
    - usually final weeks and months but could be months or years
    - includes people with:
        - advanced, progressive, incurable conditions

        - general frailty and coexisting conditions that mean they are at increased risk of dying within the next 12 months
        - existing conditions if they are at risk of dying from a sudden acute crisis in their condition
        - life-threatening acute conditions caused by sudden catastrophic events
- identification of adults approaching the end of life
    - Gold Standards Framework Proactive Identification Guidance
    - the AMBER care bundle
    - the Supportive and Palliative care Indicators Tool
    - frailty reviews
    - at the time of moving from proactive to palliative care
- holistic needs assessment
    - should include spiritual, health and social care needs
    - identifies concerns and problems so that support can be provided
- ReSPECT
    - background
        - stands for Recommended Summary Plan for Emergency Care and Treatment
        - summary of personalised recommendations for a person's clinical care in a future emergency in which they do not have capacity to make or express choices
        - intended to respect both patient preference and clinical judgement
        - includes whether CPR should be attempted
        - plan should stay with the patient
    - elements
        - patient details
        - about the person and their health
        - preferences for care
        - clinical recommendations for care
        - CPR decision
        - decisions about mental capacity
        - clinician details
        - emergency contact details and those involved in making the plan
        - review details
    - process of the ReSPECT conversation
        - discussing and reaching a shared understanding of the person's current state of health and how it may change in the foreseeable future
        - identifying the person's preferences for and goals of care in the event of a future emergency
        - using that to record an agreed focus of care – either moving towards life-sustaining treatments or more towards prioritising comfort over efforts to sustain life
        - making and recording shared decisions about specific types of care and realistic treatment that they would want considered, or that they would not want, and explaining sensitively advance decisions about treatments that clearly would not work in their situation
        - making and recording a shared decision about whether or not CPR is recommended

EIC4 dementia – cognitive impairment

- definition
    - a syndrome caused by a number of brain disorders causing memory loss, decline in some other aspect of cognition and difficulties with activities of daily living
    - three groups of symptoms
        - cognitive impairment
            - difficulties with memory, language, attention, thinking, orientation, calculation and problem-solving
        - psychiatric or behavioural disturbances
            - changes in personality,, emotional control and social behaviour, depression, agitation, hallucinations and delusions
        - difficulties with activities of daily living, such as driving, shopping, eating and dressing
    - there is progressive decline from a previous higher level of functioning and consciousness is not clouded
    - long term memory may remain intact
- common causes of dementia
    - Alzheimer's disease
        - about 50%
        - degeneration of the cerebral cortex with cortical atrophy, neurofibrillary tangles, amyloid plaque formation and reduction in acetylcholine production from affected neurons
    - vascular dementia
        - about 25%
        - brain damage due to cerebrovascular disease e.g. major stroke, multiple unrecognised smaller strokes or chronic changes in smaller vessels
    - dementia with Lewy bodies
        - about 15%
        - deposition of abnormal protein within neurons in the brain stem and neocortex
    - frontotemporal dementia
        - <5%
        - specific degeneration/atrophy of the frontal and temporal lobes
        - in Pick's disease, protein tangles are seen histologically
    - mixed dementia
    - Parkinson's disease
    - potentially treatable dementias
        - <5%
        - includes:
            - substance misuse
            - hypothyroidism
            - space-occupying intracranial lesions
            - normal pressure hydrocephalus
            - syphilis
            - vitamin B12 deficiency
            - folate deficiency
            - pellagra

    - genetic causes of dementia
- diagnosis
    - elements involved include:
        - full history and physical examination
        - specific attention to:
            - attention and concentration ability
            - orientation – time, place and person
            - memory – short- and long-term
            - praxis – whether they can get dressed, lay a table etc.
            - language function
            - executive function – problem solving etc.
            - psychiatric features – depression, anxiety, psychotic symptoms
        - medication review
        - formal screen for cognitive impairment
            - Mini Mental State Examination (MMSE)
            - 6 item cognitive impairment test (6CIT)
            - General Practitioner Assessment of Cognition (GPCOG)
            - 7 minute screen (7MS)
        - exclusion of other reversible causes
    - diagnostic criteria include:
        - cognitive or behavioural symptoms which:
            - affect ability to function in normal activities
            - represent a decline from a previous level of function
            - cannot be explained by delirium or other major psychiatric disorder
            - have been established by history taking from patient and informant, and formal cognitive assessment
        - involve impairment of at least two of the following domains:
            - ability to acquire and remember new information
            - judgement, ability to reason or handle complex tasks
            - visuospacial ability
            - language functions
            - personality and behaviour
- investigations
    - to look for any treatable/reversible cause
        - bloods
            - FBC, ESR/CRP, U&E, LFT, glucose, Ca^{2+}, TFT, B12 +/- homocysteine, folate
        - consider MSU
        - VDRL if risk factors for syphilis present
        - blood cultures if indicated
        - consider CXR/MRI
        - consider psychometric testing
        - CSF if Creutzfeldt-Jakob or other rapidly progressive dementia suspected
        - specialist assessment to determine the subtype of dementia
- management
    - NHS England has a ‘Well pathway for dementia’ document
        - sets out what good quality assessment, diagnosis and care look like

    - person-centred care
        - patients with dementia should not be discriminated against when considering treatment options for other conditions
        - early discussions regarding advance planning
            - advance statements, lasting power of attorney, preferred place of care
        - memory assessment service as single point of referral for all patients with suspected diagnosis of dementia
        - valid consent for treatment where possible
            - use of advocacy and voluntary organisations where possible
            - use of the Mental Capacity Act where needed
        - carers should have an assessment of needs and be offered support
        - joint planning of services by health and social care managers
        - following diagnosis the patient should be given written information about:
            - the symptoms and signs of dementia
            - course and prognosis
            - treatments
            - local care and support services
            - support groups
            - sources of financial and legal advice and advocacy
            - medico-legal issues, including driving
                - there is a legal obligation for the patient to inform the DVLA
                    - some patients may be able to continue driving with annual reviews
                    - drivers of HGV or passenger-carrying vehicles would have their licence revoked
            - local information sources, including libraries and voluntary organisations
- management
    - non-pharmacological
        - cognitive stimulation programmes
        - multisensory stimulation
        - music therapy
        - art therapy
        - dancing
        - massage
        - aromatherapy
        - structured exercise programme
        - animal-assisted therapy
    - community and hospital care
        - the aim should be for care in the community wherever possible
    - factors that may exacerbate challenging, violent or aggressive behaviour
        - overcrowding
        - lack of privacy
        - boredom or lack of activity
        - poor communication
        - conflict

        - weak clinical leadership in care home settings
    - pharmacological management
        - acetylcholinesterase inhibitors
            - donepezil, galantamine, rivastigmine
            - considered in patients with mild or moderate Alzheimer's disease
                - should not be used for non-Alzheimer's dementia
            - only started by dementia specialists after discussion with family and carers
            - started at low doses and titrated
            - cholinergic side-effects
            - should only be continued for as long as they are having a worthwhile effect on cognitive, global, functional or behavioural symptoms and should be regularly reviewed
        - N-methyl-D-aspartate (NMDA) antagonists
            - memantine
            - recommended by NICE as a second-line option for moderate Alzheimer's disease where AChE inhibitors are not tolerated or are contra-indicated or in the treatment of severe Alzheimer's disease
            - can be used in addition to an AChE inhibitor for moderate or severe dementia
        - pharmacological treatment of associated non-cognitive problems
            - antidepressants
                - avoid tricyclic antidepressants and other anticholinergics – may have an adverse effect on cognition
            - antipsychotics
                - should be avoided where possible in Alzheimer's disease, vascular dementia or mixed dementias
                - where required for psychotic features or agitation:
                    - discussion of risks (e.g. sedation, risk of stroke, worsening cognition)
                - treatment under specialist advice where possible
                - regular monitoring of effects
                - use lowest dose and titrate slowly upwards
                - treatment should be time-limited
                - risperidone should be considered as a first
                - severe sensitivity reactions are more likely in those with Parkinson's disease or Lewy Body dementia and they should be avoided where possible
            - urgent behavioural management
                - where aggression, violence or agitation pose a threat to safety and non-pharmacological measures have failed
                - oral medication where possible, IM as second line
                - if IM treatment required, consider lorazepam, haloperidol or olanzapine
                - effects should be closely monitored
    - palliative and end of life care
        - physical, psychological, social and spiritual support should be offered

        - access to palliative care services should be available
        - continue oral nutrition for as long as possible
            - PEG feeding may be considered for transient dysphagia but is not recommended in severe dementia as there is no evidence of increased survival or reduced complications
            - decisions to withhold nutritional support should be taken within a legal and ethical framework
        - fever may be managed with antipyretics and mechanical cooling
        - palliative antibiotics may be given after individual assessment
        - resuscitation is unlikely to succeed in patients with severe dementia and the multidisciplinary team and carers should be involved in these decisions where there is no advance plan
- prevention
    - genetic screening for families of those with a suspected genetic cause
    - modification of risk factors in middle age:
        - smoking
        - alcohol consumption
        - obesity
        - other cerebrovascular disease risk factors such as hypertension and hypercholesterolaemia

EIC5 fragility fractures

- background
    - fractures that result from mechanical forces that would not ordinarily result in fracture
        - WHO quantifies as fall from standing height or less
    - risk factors include:
        - osteoporosis
        - advancing age
        - conditions affecting bone strength (e.g. acromegaly or osteogenesis imperfecta)
        - predisposition to falls due to loss of balance or poor muscle strength
    - over 200,000 fractures per year in the UK caused by osteoporosis
        - expected to double over next 50 years
        - 1 in 3 women and 1 in 5 men will have one or more osteoporotic fractures in their lifetime
    - vertebral fragility fractures may occur following bending or sneezing or lifting
- risk factors
    - reduced bone mass density
    - increasing age
    - female gender
    - low body mass and anorexia nervosa
    - parental history of hip fracture
    - past history of fragility fracture
        - especially wrist, hip, spinal
    - corticosteroid therapy
        - current treatment at any dose orally for three months or more

    - Cushing's syndrome
    - alcohol intake of >3 unites/day
    - smoking
    - ethnicity
        - higher risk in Caucasians
    - other causes of abnormal bone
    - falls and conditions increasing the risk of falls
        - visual impairment
        - lack of neuromuscular co-ordination or strength
        - cognitive impairment
        - sedative medication and alcohol
    - secondary causes of osteoporosis
        - rheumatoid arthritis and other inflammatory arthropathies
        - prolonged immobilisation or very sedentary lifestyle
        - primary hypogonadism
        - primary hyperparathyroidism
        - hyperthyroidism
        - post-transplantation
        - CKD
        - gastrointestinal disease (e.g. Crohn's ulcerative colitis, coeliac disease)
        - untreated premature menopause (<45 years) or prolonged secondary amenorrhoea
        - type 1 and type 2 diabetes
        - chronic liver disease
        - COPD
- presentation
    - most common sites are:
        - vertebrae
        - hip
        - distal radius
    - other affected sites include:
        - pelvis
        - ribs
        - arm
        - shoulder
    - vertebral fractures may not be immediately noticed
        - reasons for underdiagnosis include:
            - may not result from a fall
            - symptoms are often attributed to another cause by patient and healthcare professionals
            - the need for spinal imaging may not be recognised where there is new back pain and risk factors for osteoporosis
            - the spine may not be sufficiently scrutinised on imaging if imaged for other indications
            - ambiguous and confusing terminology may be used in reporting

                - the clinician may regard the finding of a vertebral fracture as incidental to the reason for the original referral and fail to recognise the clinical importance
- effects of compression fractures
    - pain and morbidity associated with high doses of analgesia
    - loss of height
    - loss of mobility
    - gastrointestinal symptoms
    - difficulty sleeping
    - symptoms of depression
- management
    - acute
        - pain relief
        - management of associated chronic disease
        - fluid management
        - fracture stabilisation
        - possible investigations prior to surgery:
            - CXR
            - ECG
            - FBC, G&S, clotting
            - U&Es
            - glucose
            - assessment of cognitive function
    - consider liaison with the medical team
    - rehabilitation and education
        - muscle strengthening exercises
        - physiotherapy
        - fall prevention measures
        - balance training
        - medication review
        - education on modifiable risk factors
    - assessment for risk of future fragility fracture
        - assessment of risk factors
        - DXA where appropriate
        - NICE guidelines suggest it is reasonable to assume a diagnosis of osteoporosis if there has been a fragility fracture and treatment should be started
    - treatment of low bone density
        - adequate calcium and vitamin D with supplementation as needed
        - high risk of future fracture (by risk calculator)
            - bisphosphonates
                - alendronate or risedronate orally daily or weekly
                - ibandronate orally monthly or IV three-monthly
                - zoledronic acid by IV infusion annually
            - denosumab
                - monoclonal antibody that reduces osteoclast activity
                - six-monthly subcutaneous injections

        - approved for post-menopausal women with increased fracture risk who cannot comply with special instructions or have intolerance for bisphosphonates
    - strontium ranelate
        - only where other treatments are not possible
        - cannot be used in current or past history of coronary heart disease, uncontrolled hypertension, peripheral arterial disease and/or cerebrovascular disease
    - raloxifene
        - selective oestrogen receptor modulator which inhibits bone resorption
        - shown to reduce vertebral fracture risk but not other types of fracture
    - teriparatide
        - recombinant fragment of parathyroid hormone prescribed in secondary care
        - may be considered for very severe osteoporosis or very high fracture risk when unable to use bisphosphonates or they have been ineffective
    - hormone replacement therapy
        - option for younger perimenopausal women who also need treatment for symptoms of menopause
- prognosis
    - varies with age, comorbidity, fracture site, other risk factors and personal circumstances
    - may result in reduced quality of life, pain and disability
    - having one fragility fracture is a significant risk factor for another

EIC6 mobility

- epidemiology
    - most common risk factors for mobility impairment:
        - older age
        - low physical activity
        - obesity
        - strength or balance impairment
        - chronic disease such as diabetes or arthritis
    - prevalence of gait and balance disorders is around 10% between the ages of 60 and 69 and 50% in those over 80
- assessment
    - history
        - patients may complain directly of problems with walking or unsteadiness
        - it is important to clarify which aspect of walking the patient finds difficult
        - ask about falls
            - when the last fall occurred
            - how frequent falls are
            - whether there are any syncope or presyncope symptoms
        - duration of problems

        - full systems review
        - assess for features of cord compression
        - full drug history
    - examination
        - pulse rate, rhythm, volume and presence or absence of carotid bruits
        - blood pressure including postural hypotension
        - cardiovascular examination
            - murmurs
        - full neurological examination
            - pyramidal, extrapyramidal, cerebellar dysfunction, peripheral neuropathy
        - check for fractures and other injuries
        - gait examination
            - symmetrical or asymmetrical
            - waddling gait
            - broad based gait
            - scissoring gait (bilateral leg spasticity)
            - ataxia
        - further testing
            - timed get up and go test
                - person gets up from chair without using arms, walks three metres, turns around, returns to the chair and sits down
                - usual walking aid should be used
                - score of 12-15 seconds or more indicates high risk of falls in older people
            - turn 180 test
                - person stands up and steps around until facing the opposite direction
                - if they take more than four steps, further assessment should be considered
- causes of difficulty in walking
    - vascular
        - cardiac
            - arrhythmias
            - hypotension
            - postural hypotension
        - neurological
            - transient ischaemic attacks
            - CVA
            - multi-infarct dementia
    - neurological
        - pyramidal disease
            - multiple sclerosis
            - cord compression
            - motor neurone disease
            - syringomyelia
            - spinal cord tumours

                - B12 deficiency
                - syphilis
            - extrapyramidal disease
                - tardive dyskinesia
                - akathisia
                - Parkinson's disease
                - Parkinsonism
            - cerebellar disease
                - cerebellar tumours
                - any ataxia (e.g. Friedreich's ataxia)
                - Wernicke's encephalopathy
            - other
                - peripheral neuropathy
                - chorea
    - orthopaedic
        - painless
            - arthrodesis of hip joints
        - painful
            - arthritides (e.g. osteoarthritis, rheumatoid arthritis)
            - spinal disease (e.g. stenosis)
            - fractures
            - foot problems (e.g. corns, bunions, ill-fitting shoes)
    - balance and co-ordination
            - Alzheimer's disease
            - labyrinthitis
            - degenerative changes in the inner ear
    - muscles
            - myopathies
    - metabolic
            - diabetes mellitus (e.g. autonomic neuropathy, foot drop)
            - thyroid disorders
    - others
        - toxins/drugs
            - antihypertensive medications
            - sedatives
            - antipsychotics
            - ethanol
            - anticonvulsants
        - psychological
            - loss of confidence, including depression
- causes of 'off legs'
    - urine or chest infections
    - dehydration
    - neurological causes
        - head injury
        - cord compression/cauda equina syndrome
    - orthopaedic causes

        - fractures
    - metabolic abnormalities
        - hyponatraemia
        - hypercalcaemia
        - hypoglycaemia
        - hyperglycaemia
    - alcohol, drugs or medications
        - including polypharmacy
    - hypoxia
- investigations
    - guided by history and examination
    - may include cerebral imaging, blood tests
- management
    - directed towards the underlying cause
    - if multifactorial a multidisciplinary approach may be appropriate
        - e.g. physiotherapist, occupational therapist, allied healthcare professionals
    - falls prevention strategies

EIC7 osteoporosis

- definition
    - progressive systemic disease characterised by reduced bone density and micro-architectural deterioration of bone tissue
        - bone is increasingly fragile and more susceptible to fracture
- bone density
    - expressed in relation to a reference population in standard deviations
        - a T score is used in relation to the young healthy population
        - a Z score compares bone density to the normal at that age
    - WHO BMD categories
        - normal
            - hip BMD greater than the lower limit of normal (T score ≥ -1)
        - low bone mass (osteopenia)
            - hip BMD between 1 and 2.5 SD below the young adult reference mean
        - osteoporosis
            - hip BMD 2.5 SD or more below the young adult reference mean
        - severe osteoporosis
            - hip BMD 2.5 SD or more below the young adult reference mean in the presence of one or more fragility fractures
- risk assessment
    - clinical risk factors
        - increasing age
        - female sex
        - low body mass index and anorexia nervosa
        - parental history of hip fracture
        - past history of fragility fracture (especially hip, wrist, spine)
        - corticosteroid therapy
        - Cushing's syndrome

        - alcohol intake of >3 units/day
        - smoking
        - falls and conditions increasing the risk of falls
    - secondary causes of osteoporosis
        - rheumatoid arthritis and other inflammatory arthropathies
        - prolonged immobilisation or very sedentary lifestyle
        - primary hypogonadism
        - treatment with aromatase inhibitors or androgen deprivation therapy
        - primary hyperparathyroidism
        - hyperthyroidism
        - post-transplantation
        - CKD
        - gastrointestinal disease (e.g. Crohn's disease, ulcerative colitis, coeliac disease)
        - untreated premature menopause (<45 years) or prolonged secondary amenorrhoea
        - type 1 diabetes
        - chronic liver disease
        - COPD
    - pharmaceutical agents
        - proton pump inhibitors
        - enzyme-inducing anticonvulsants
        - long-term depot medroxyprogesterone acetate
        - long-term antidepressants
        - thiazolidinediones (anti-diabetic therapy)
- risk assessment guidelines
    - NICE recommends fracture risk assessment in:
        - all women 65 and over
        - all men 75 and over
        - women under 65 and men under 75 in the presence of risk factors, e.g.:
            - previous fragility fractures
            - current use or frequent use of oral or systemic glucocorticoids
            - history of falls
            - family history of hip fracture
            - causes of secondary osteoporosis
            - low body mass index
            - smoking
            - >14 units/week of alcohol
- assessment of fracture risk (primary care)
    - 10-year risk assessment via
        - FRAX risk assessment
        - QFracture calculator
- interventions
    - patients at increased risk of fracture should be offered a bone-preserving agent
    - primary prevention
        - encouragement to take adequate calcium

        - supplementation with calcium and vitamin D where intake may be suboptimal
    - healthy diet with '5 a day' of fruit and vegetables
    - reduced salt and phosphate intake
    - moderation of alcohol
    - anti-smoking advice
    - encouragement of exercise
        - traditional weight-bearing exercise
        - exercise involving pulling forces on entheses (tendon insertions) of long bones

EIC8 pharmacology considerations in the older patient

- background
    - 45% of prescriptions in the UK are dispensed to patients over 65
    - taking multiple agents increase the risk of suffering adverse drug reactions and interactions
    - pharmacokinetics and pharmacodynamics may be altered by normal ageing or disease
        - clinical trials rarely recruit patients >65 years
- caution in prescribing
    - careful attention to distinguishing between symptoms due to normal ageing and those due to specific treatable diseases
    - careful selection of appropriate agents
- guidance for prescribing in older adults
    - balance the potential harm and benefits of a given agent
    - conduct a regular review of older patients' prescriptions and assess the risk/benefit balance in an ongoing fashion
        - medicines appearing to have no benefit or to have unacceptable adverse effects should be stopped
    - consider non-pharmacological treatments for common symptoms such as dizziness, insomnia and headache
    - psychological factors such as recent bereavement and social isolation should be considered and addressed
    - when prescribing prophylactic medications, consider whether they are appropriate in the context of the whole person and their comorbidities, the risks of taking medication, the likelihood of compliance and the population from which the original evidence of effectiveness was identified
    - older patients should not be denied preventative pharmacological agents such as warfarin and statins but their use should be carefully considered
- use of appropriate formulations
    - attention to swallowing problems
    - tablets that remain in the mouth or oesophagus for long periods may cause ulceration
    - consider liquid formulations or give advice about taking tablets with plenty of water whilst sitting upright
- avoid symptomatic prescribing

    - avoid attempting to treat a normal aspect of ageing such as alteration in the sleep-wake cycle
- consider effects of non-prescribed medication
    - ask about OTC or complementary preparations which could interact with prescribed medications
    - consider use of previously prescribed agents or agents prescribed for other people
    - find out what the patient understands about what they are taking, how and when they should be taking them
- pharmacological differences between younger and older patients
    - pharmacodynamics
        - the CNS is more sensitive to agents such as antipsychotics, opioids, benzodiazepines, anti-Parkinsonian agents
        - drugs with toxic gastrointestinal side effects such as NSAID and opioids should be used with caution
    - particular care with CNS-active drugs that affect balance, wakefulness, motor function and perception in the older patient prone to falls
- pharmacokinetics
    - the most important alteration in the elderly is reduction in renal clearance
        - accumulation and adverse events can result
        - particular care should be taken with drugs known to cause nephrotoxicity (e.g. NSAIDs, ACEi, aminoglycosides)
        - renally excreted drugs with a narrow therapeutic index such as digoxin should be used cautiously and at low doses
            - consider checking drug levels if toxicity suspected
    - other pharmacokinetic considerations in the elderly include:
        - drug absorption changes little, but there may be a significant increase in the absorption of levodopa
        - bioavailability may be increased for drugs which are extensively metabolised in the liver (e.g. propranolol, verapamil, many psychotropics) due to loss of first pass metabolism
            - interactions may occur with multiple therapy
            - drugs cleared by the liver should be used with extreme caution in older patients with hepatic impairment
        - the apparent volume of drug distribution is altered due to changes in lean body mass
            - there may be a reduction in drug distribution volume for some water-soluble drugs (e.g. digoxin) and an increase for lipophilic drugs (e.g. diazepam)
        - protein binding may be altered but is not usually a problem associated with normal ageing
            - disease which reduces albumin levels is more common in the elderly and should be considered when prescribing heavily protein-bound drugs such as warfarin and sulphonylureas
        - long-term use of thiazide diuretics causes only a small change in body potassium in the middle-aged but is a major cause of deficiency in the elderly due to reduced dietary intake
- adverse drug reactions in older patients

    - confusion can be caused by virtually any drug
    - other common side effects are constipation, dizziness, dry mouth, blurred vision
    - falls are often associated with medication
        - e.g. benzodiazepines, antidepressants, antipsychotics, antiepileptics, antihypertensives
- side effects of specific drug classes
    - NSAIDs
        - gastrointestinal bleeding is more common and has more serious consequences in older patients
        - NSAIDs can worsen heart failure or aggravate impaired renal function
        - best avoided for simple pain relief
            - if paracetamol is insufficient, a low dose NSAID can be tried with PPI cover, or consider a low dose opioid
        - consider complementary therapies such as acupuncture to help with pain
        - co-prescription of NSAIDs and ACEis can be disastrous, causing significant renal impairment
    - hypnotics
        - hypnotics with long half-lives can cause daytime drowsiness, unsteadiness from impaired balance and confusion
        - short-acting ones can also be problematic and should only be used for short periods if essential
        - avoid in patients prone to falls or dizziness
        - sleep hygiene and non-pharmaceutical measures should be tired for insomnia
    - diuretics
        - should not be used for chronic treatment of gravitational oedema
            - measures such as leg-raising, increased walking, leg exercises and graduated compression stockings are often sufficient
        - when used to treat hypertension or cardiac failure they should be reviewed regularly with an assessment of hydration status and U&Es
        - withdrawal of diuretics requires careful monitoring of effects such as decompensation of heart failure or significant rise in blood pressure
    - digoxin
        - daily maintenance dose should be 125 micrograms in the elderly, reduced to 62.5 microgram in renal impairment
    - drugs that cause bone marrow suppression
        - e.g. co-trimoxazole, chloramphenicol
        - should only be used if there is no alternative
    - anticoagulants and antiplatelets
        - consider gastrointestinal bleeding risk and contraindications such as peptic ulcers
        - warfarin should only be prescribed if patients have a full understanding of why it is being taken, its dangers, correct daily dosing and timing and the importance of regular INR monitoring
    - antidepressants
        - tricyclics commonly cause postural hypotension and confusion in the elderly

        - serotonergic medications may cause serotonin syndrome and agitation, which may be difficult to distinguish from some of the symptoms of depression
    - diabetic medication
        - long acting oral hypoglycaemics should be avoided – significant risk of hypoglycaemia when used in the older patient
        - tight diabetic control must be balanced against potentially catastrophic events precipitated by hypoglycaemia, particularly in those who live alone, have a poor understanding of diabetes self-management or experience few waning symptoms of hypoglycaemia
- sensible prescribing in older patients
    - is It needed?
        - consider pros, cons and the evidence base in the older patient
    - limit the range of drugs used in older patients
        - have a small formulary for older patients and be aware of the indications, contra-indications and potential side effects
    - start low, go slow
        - often start at half the normal adult dose
    - keep it simple
        - use drug regimens with the lowest number of different agents and with dosing intervals of once or twice a day
        - avoid complicated or multiple dosing regimens where possible
        - avoid polypharmacy
        - balance holistic considerations with an evidence based approach
    - make it clear
        - avoid imprecise prescribing such as 'as directed' – give full dose, frequency and route
        - use dosette boxes or other aids where necessary
    - review regularly
        - GP review to assess whether medication should be continued long-term
    - use of teams and support
        - interdisciplinary approaches to prescribing, dispensing and monitoring of medicines
        - use of colleagues and clinical pharmacists

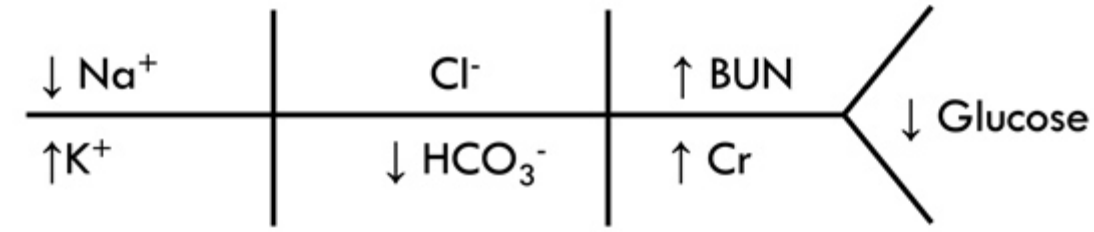
ENDOCRINOLOGY

EnP1 Addisonian crisis

- definition
    - no established formal definition
    - reasonable definition:
        - acute deterioration with absolute hypotension (systolic <100) or relative hypotension (drop of >20 mmHg)
        - resolution within 1-2 hours of IV steroid administration
    - steroids can be given if in doubt and may be diagnostic

    - consider adrenal crisis in any patient who appears to be septic but no source of infection found
- physiology
    - cortisol and aldosterone deficiency affects most organs and tissues
    - impaired suppression of inflammation
        - cortisol inhibits pro-inflammatory cytokines normally
        - release of inhibition increases systemic inflammation
        - clinical manifestations include fever, malaise, anorexia
    - haemodynamic effects
        - cortisol normally functions synergistically with catecholamines to cause vasoconstriction
    - metabolic effects
        - cortisol is generally catabolic, stimulating the production of various fuels in times of stress
            - gluconeogenesis in the liver
            - production of free fatty acids
            - release of amino acids
        - adrenal crisis can lead to low levels of these fuels, particularly clinically detectable hypoglycaemia
    - renal effects
        - aldosterone is deficient only in cases involving the entire adrenal gland (e.g. Addison's disease)
        - aldosterone deficiency causes wasting of sodium with retention of positively charged ions (K+ and H+)
            - leads to hyperkalaemia and non-ion gap metabolic acidosis
- epidemiology
    - chronic adrenal insufficiency + stressor
        - common causes of chronic adrenal insufficiency:
            - Addison's disease
            - chronic steroid therapy (most common cause)
        - adrenal crisis precipitated by acute stressors such as:
            - infection
                - especially gastroenteritis, but adrenal crisis symptoms may mimic gastroenteritis
            - trauma/surgery
            - volume depletion
            - pregnancy
            - physiological stress, exercise
            - reduced steroid dose
                - non-adherence
                - tapering of doses
            - initiation of drugs inducing cytochrome P-450 3A4 (increases hydrocortisone metabolism):
                - carbamazepine
                - etomidate
                - ketoconazole/fluconazole
                - phenytoin

        - rifampin
        - St John's wort
        - thyroid hormone
    - acute adrenal insufficiency (less common)
        - usually in the context of another illness:
            - Waterhouse-Friedrichson syndrome
                - adrenal infarction due to DIC
            - pituitary apoplexy
                - pituitary infarction, often post-partum or due to DIC
            - cancer patients receiving checkpoint-inhibitor immunotherapy
                - may experience acute failure of the adrenal and/or pituitary gland
- signs and symptoms of adrenal crisis
    - main findings
        - hypotension, vasodilatory shock
            - often refractory to fluid and vasopressors
        - nausea/vomiting, abdominal pain/tenderness
        - fever
    - other findings
        - delirium
        - features of the trigger of the crisis (e.g. trauma, surgery)
    - clues for patients with chronic adrenal insufficiency
        - cutaneous hyperpigmentation or vitiligo
        - history of chronic fatigue, anorexia, vomiting and weight loss
- investigations



Textbook changes in chemistry panel due to adrenal insufficiency.
Of course, "textbook" means "rarely seen in actual clinical practice."
The Internet Book of Critical Care, by @PulmCrit

https://emcrit.org/ibcc/adrenal-crisis/

    - electrolyte abnormalities due to mineralocorticoid deficiency
        - seen in primary adrenal insufficiency (e.g. Addison's) but not secondary (e.g. pituitary dysfunction)
        - causes type 4 renal tubular acidosis
            - hyperkalaemia
            - non-anion gap metabolic acidosis
    - electrolyte abnormalities seen with any type of adrenal insufficiency:
        - hyponatraemia
        - hypoglycaemia
        - hypercalcaemia

    - absence of electrolyte abnormality does **not** exclude crisis
    - eosinophilia
        - normally, physiological stress stimulates cortisol production which decreases eosinophil counts
        - normal or increased eosinophil counts in a critically ill patient raise the possibility of adrenal crisis
    - further testing if adrenal crisis suspected
        - random cortisol level
            - suggestive if inappropriately low
            - excludes adrenal crisis if >20 ug/dL
        - ACTH stimulation test
            - Synacthen given
            - cortisol measured at baseline and after 60 minutes
            - if it does not rise above 20-25 ug/dL, adrenal insufficiency is concerned
            - can be done if patient if on dexamethasone but not on any other steroids
- management
    - investigation and treatment of trigger
        - cultures and empirical antibiotics appropriate if suspicion of infection
    - steroid
        - for known adrenal sufficiency#
            - 100mg IV hydrocortisone STAT
                - then 50mg IV every 6 hours for maintenance
            - can be tapered to baseline dose once patient recovers
        - for suspected adrenal insufficiency
            - dexamethasone 4-6mg IV once
                - does not interfere with measurement of cortisol levels
                - does not have mineralocorticoid effect but generally not important
                    - consider fludrocortisone 0.2mg PO if hyperkalaemia
            - test for adrenal insufficiency
    - resuscitation
        - volume replacement based on haemodynamic assessment
            - often depleted due to poor oral intake and vomiting
        - consider vasopressors early
    - re-evaluation
        - should improve rapidly with steroid and resuscitation
        - failure to improve warrants further investigation, repeat examination, consider imaging
- prevention
    - patients with known adrenal insufficiency should increase steroid dose when undergoing physiological stress to prevent crisis
        - patients with severe infection, surgery etc should receive 50mg hydrocortisone IV QDS
        - smaller increases in usual steroid dose may be sufficient for lesser degrees of stress

EnP2 hyperglycaemia

- control of blood glucose
    - normal range is 4-6 mmol/L in the non-diabetic
        - rises after consumption of carbohydrate
    - glucose regulation can be divided into:
        - short-term
            - regulation via secretion or inhibition of insulin and glucagon from the pancreatic islets
        - long-term
            - regulation via both neuronal and hormonal mechanisms
    - hormonal mechanisms
        - short-term
            - glucose levels are sensed directly in the pancreas and result in insulin release when the glucose is >5.6 mmol/L
            - pancreatic beta cells respond directly to glucose by secreting insulin in a biphasic fashion
                - initial rapid increase in release
                - prolonged slow increase in release
- organ effects
    - glucose levels are influenced by the:
        - liver
            - insulin and glucagon act on the liver to continually adjust the relative rates of glycogenolysis and glycogenesis, allowing it to function as an effective buffer of blood glucose
            - hepatic disease significantly affects this, resulting in fluctuating blood glucose
        - kidney
            - transient glycosuria may be seen as hyperglycaemia decreases renal absorption of glucose
- stress-induced hyperglycaemia
    - background
        - transient hyperglycaemia associated with acute illness
        - resolves with resolution of the illness
        - marker of disease severity but does not imply causation
        - diagnosis reserved for patients without prior evidence of diabetes
    - mechanisms
        - high hepatic glucose output via gluconeogenesis driven by glucagon, adrenaline, cortisol, TNF alpha
        - insulin resistance
        - effects of therapies – TPN, enteral feed, steroids, vasopressors
        - underlying illness
    - clinical implications
        - increased mortality, length of stay, infections, more overall complications
        - unknown whether it causes harm or is a marker of illness severity
        - difficult to distinguish from other causes of hyperglycaemia
    - management
        - insulin therapy with conservative glucose targets in the critically unwell

        - control precipitant if possible
        - frequent blood glucose monitoring
        - avoid hypoglycaemia
        - probably reasonable to aim for glucose <10mmol/L

EnP3 hypoglycaemia

- background
    - no universally accepted definition
    - clinically significant hypoglycaemia is confirmed by the Whipple triad:
        - presence of symptoms consistent with hypoglycaemia
        - low serum glucose level
        - resolution of symptoms and signs of hypoglycaemia with the administration of glucose
    - clinical hypoglycaemia is defined as a blood glucose low enough to cause symptoms or signs (including brain impairment) or both
        - for most people it occurs at levels less than 2.8-3.3 mmol/L
    - severe hypoglycaemia is defined as an event requiring the assistance of another person to actively administer carbohydrate, glucagon or other resuscitative measures
    - relative hypoglycaemia occurs when a person with diabetes reports hypoglycaemic symptoms but the blood glucose remains above 3.8 mmol/L – this still requires treatment
- causes
    - known diabetics (most common)
        - hypoglycaemic agents
            - sulphonylureas
            - insulin
        - decreased glucose delivery
            - missed meals
            - overnight fasting
        - increased glucose utilisation
            - exercise
        - reduced endogenous glucose production
            - alcohol ingestion
        - increased insulin sensitivity
            - weight loss
            - increase in activity
        - reduced insulin clearance
            - renal failure
    - no history of diabetes (rare)
        - exogenous drugs
            - insulin
            - oral hypoglycaemics
            - ethanol intoxication
            - quinine
            - chloroquine
            - beta blocker overdose

                - valproate overdose
                - salicylate overdose
                - pentamidine
            - pituitary insufficiency
            - post-prandial hypoglycaemia
                - e.g. 'late dumping' after gastric surgery
            - liver disease
                - e.g. hepatocellular carcinoma, hepatitis, rare genetic defects
            - Addison's disease
            - islet cell tumours
                - e.g. insulinomas
            - immune hypoglycaemia
                - e.g. anti-insulin receptor antibodies in Hodgkin's disease
            - infection
                - severe sepsis
                - malaria
            - non-pancreatic neoplasms
                - fibromas, sarcomas, mesotheliomas, small cell carcinomas that produce IGF-2
                - extensive metastases that overwhelm the body's ability to produce glucose
            - nesidioblastosis or noninsulinoma pancreatogenous hypoglycaemia (NIPH) – islet cell hyperplasia which can be congenital or acquired, e.g. post gastric surgery
            - starvation and malnutrition
            - hypothyroidism (myxoedema coma)
        - pseudohypoglycaemia
            - delayed measurement of a sample in the presence of leucocytosis, thrombocytosis or erythrocytosis
- hormonal control
    - low glucose levels stimulate secretion of glucagon from the pancreas
        - typically less important than the effect of insulin unless in situations of starvation or severe psychological stress
    - long term
        - sustained hypoglycaemia increases fat utilisation and decreases glucose utilisation, limiting further drops in glucose, via stimulating release of GH and cortisol
- neuronal mechanisms
    - hypoglycaemia directly stimulates the hypothalamus, causing:
        - increased sympathetic nervous system tone
        - adrenaline release in turn stimulates hepatic glucose release
- physiological responses to hypoglycaemia
    - 4.6 mmol/L
        - insulin secretion inhibited
    - 3.8 mmol/L
        - autonomic dysfunction
        - glucagon, adrenaline and GH secretion

- 2.8 mmol/L
  - CNS dysfunction
- 2.2 mmol/L
  - lethargy, coma
- 1.7 mmol/L
  - convulsions
- 0.6 mmol/L
  - permanent brain damage, death

EnC1 adrenal disorders

- adrenal insufficiency
  - background
    - primary (Addison's)
      - destruction of >90% of adrenal glands
      - rare
      - causes:
        - autoimmune destruction
        - haemorrhage
        - tumour (breast, melanoma)
        - infection (TB, HIV, meningococcaemia, purpura fulminans)
        - inflammatory process
    - secondary
      - insufficient production of ACTH
      - rare
      - mineralocorticoid function intact
      - causes:
        - destruction or dysfunction of the pituitary
    - tertiary/iatrogenic/relative
      - suppression of HPA axis over time
      - most common
      - cause:
        - administration of exogenous glucocorticoids
      - mechanism:
        - chronic ACTH suppression leads to adrenal atrophy
  - chronic adrenal insufficiency
    - general
      - weight loss
      - arthralgia
      - myalgia
    - CNS
      - fatigue
      - anorexia
      - mood change
    - CVS
      - postural hypotension
      - syncope
      - salt craving

        - skin
            - pigmentation
            - vitiligo
        - electrolytes
            - hypoglycaemia
            - hyponatraemia
            - hyperkalaemia
            - increased urea
    - adrenal crisis
        - causes:
            - concurrent illness
            - surgery
            - failure to take medications
        - signs and symptoms
            - GI
                - abdominal pain
                - vomiting
                - diarrhoea
            - CVS
                - dehydration
                - hypotension
                - refractory shock
                - poor response to intropes/pressors
            - fever
            - confusion
    - investigations
        - diagnosis
            - plasma cortisol level <80 mmol/L
            - short synacthen test 250 mcg - normal response is cortisol >525 mmol/L
        - other
            - low glucose
            - low sodium
            - hypo-osmolarity
            - raised potassium
            - raised urea and creatinine
            - raised calcium (primary only)
            - eosinophilia
    - management
        - fluid resuscitation
        - reversal of electrolyte abnormalities
        - high dose hydrocortisone
            - 100mg IV QDS
        - mineralocorticoid replacement
            - fludrocortisone PO 0.1mg QDS
            - not required acutely as there is a mineralocorticoid effect with >50mg hydrocortisone

- Cushing's syndrome
    - background
        - caused by prolonged exposure to elevated levels of exogenous or endogenous glucocorticoids
        - presentation may be obvious or subtle
        - there is significant morbidity and mortality so early diagnosis is important
        - iatrogenic causes are the most common
    - risk factors
        - Cushing's syndrome due to an adrenal or pituitary tumour is more common in females (5:1)
        - peak incidence of Cushing's syndrome caused by an adrenal or pituitary adenoma is between 25 and 40 years
        - ectopic ACTH production due to lung cancer occurs later in life
    - causes
        - exogenous Cushing's syndrome
            - glucocorticoid use
        - endogenous Cushing's syndrome
            - corticotropin-dependent (80-85% of cases)
                - 80% due to pituitary adenomas (Cushing's disease)
                - 20% due to ectopic corticotropin syndrome
                    - usually due to small cell carcinoma of the lung and bronchial carcinoid tumours
                    - may occur with almost any endocrine tumour (e.g. phaeochromocytoma, pancreatic neuroendocrine tumours, medullary thyroid cancer, gut carcinoid)
            - corticotropin-independent
                - usually due to a unilateral tumour
                    - adrenal adenoma in 60%
                    - adrenal carcinoma in 40%
                - very rare adrenal causes
                    - corticotropin dependent macronodular adrenal hyperplasia
                    - primary pigmented nodular adrenal disease
                    - McCune-Albright syndrome
    - presentation
        - truncal obesity, supraclavicular fat pads, buffalo hump, weight gain
        - facial fullness, moon facies, facial plethora
        - proximal muscle wasting and weakness
        - diabetes and impaired glucose tolerance
        - gonadal dysfunction, reduced libido
        - hypertension
        - nephrolithiasis
        - skin atrophy, purple striae, easy bruising, hirsutism, acne, pigmentation (with ACTH-dependent causes)
        - psychological problems: depression, cognitive dysfunction, emotional lability
        - osteopenia, osteoporosis
        - oedema
        - irregular menses

        - thirst, polydipsia, polyuria
        - impaired immune function – increased infections, difficulty with wound healing
        - growth restriction in children
        - with ACTH-producing pituitary tumour: headache, visual problems, galactorrhoea
        - destruction of the anterior pituitary may cause hypothyroidism and amenorrhoea
    - differentials
        - chronic severe anxiety and depression
        - prolonged excess alcohol consumption – can cause a Cushingoid appearance
        - obesity
        - poorly controlled diabetes
        - HIV infection
    - investigations
        - usually done in secondary care with no single test being perfect
        - acute intercurrent illness can cause false positive results
        - investigations include:
            - FBC – raised WCC
            - electrolytes and acid base balance – may have hypokalaemia and metabolic alkalosis
            - 24-hour urinary free cortisol
                - ideally three collections
                - confidently diagnosed if two or more collections measure cortisol excretion as more than three times the upper normal limit
                - may need to be repeated if secretion varies more than 10% between collections
                - false positives may occur in:
                    - pregnancy
                    - anorexia
                    - exercise
                    - psychoses
                    - alcohol
                    - alcohol withdrawal
            - low dose dexamethasone suppression test
                - useful if 24 urine cannot be collected
                - 1mg dexamethasone ingested at 11pm and serum cortisol measured at 8am the next morning
                - false positive and false negative results can occur
            - midnight cortisol levels
                - taken between 11pm and 1am
                - demonstrates loss of normal diurnal variation in cortisol
                - blood is taken from an indwelling cannula with the patient relaxed
                - inconvenient but reliable test
                - late night salivary cortisol testing may be done

- dexamethasone suppressed corticotropin-releasing hormone test
    - modification of low dose dexamethasone suppression test
- investigation of the cause
    - plasma ACTH
        - diagnostic if undetectable with an elevated serum cortisol level
    - inferior petrosal sinus sampling
    - pituitary MRI
    - CT chest and abdomen
        - for suspected adrenal tumours/ectopic ACTH
    - plasma CRH

- management
    - tumour resection for definitive management
        - risk can be increased due to poor wound healing and metabolic consequences
        - pituitary tumours
            - trans-sphenoidal microsurgery
            - radiation therapy may be used as an adjunct
            - bilateral adrenalectomy may be required
        - adrenocortical tumours
            - surgical removal
        - ectopic ACTH production
            - removal of neoplastic tissue
            - metastatic spread makes a surgical cure unlikely or impossible
            - bilateral adrenalectomy may be required
    - drug therapy
        - can be used to stabilise patient whilst awaiting surgery
            - cortisol levels must be controlled before surgery or radiotherapy if possible
        - also used for acutely ill patients, patients with unknown tumour location or unresectable lesions, patients unfit for surgery and patients with persistently raised postoperative glucocorticoid levels
        - drugs include:
            - metyrapone
            - ketoconazole
            - mitotane
        - may be used long term
        - combination therapy may be used
        - etomidate can be used for acute control of severe hypercortisolaemia
- complications
    - metabolic syndrome
    - hypertension
    - impaired glucose tolerance and diabetes
    - obesity
    - hyperlipidaemia

        - thrombophilia
        - osteoporosis
        - perforated viscera
        - impaired immunity, including opportunistic fungal infections
        - Nelson's syndrome may follow bilateral adrenalectomy
    - prognosis
        - causes of death in untreated Cushing's syndrome include:
            - vascular disease (MI/stroke)
            - uncontrolled diabetes
            - infections
        - course is usually chronic with cyclic exacerbations and rare remissions
        - prognosis may be favourable with surgery
        - the rare adrenocortical carcinomas have a 5 year survival rate of 30% or less

EnC2 diabetic ketoacidosis

- diagnostic criteria
    - pH <7.3
    - ketosis
    - HCO_3^- <15 mmol/L
        - due to high anion gap metabolic acidosis
    - hyperglycaemia
        - may be mild and can be euglycaemic
- pathogenesis
    - increased glucagon, cortisol, catecholamines, GH
    - decreased insulin
        - hyperglycaemia
        - hyperosmolality and glycosuria
        - electrolyte loss
        - ketone production from metabolism of triglycerides
        - acidosis
- history
    - symptoms
        - dry
        - abdominal pain
        - polyuria
        - weight loss
        - coma
    - risk factors
        - non-compliance
        - illness
        - newly diagnosed
    - normal insulin regime
    - diabetic control
    - previous DKA/admissions
    - previous ICU admissions
    - symptoms of precipitants
- examination

    - volume assessment
    - signs of cause (e.g. infection)
    - GCS
    - work of breathing
- investigations
    - ABG/VBG
    - electrolytes
    - osmolality
    - ketones
    - pregnancy test
    - FBC
    - ECG
    - CXR if indicated
- management
    - goals
        - establish precipitant and treat
        - assess severity of metabolic derangement
        - cautious fluid resuscitation with replacement of body water
        - provision of insulin
        - replacement of electrolytes
    - resuscitate
        - intubation for airway protection if required
        - oxygen as required
        - IV access
        - fluid boluses (20ml/kg sodium chloride or Hartmann's)
        - urinary catheter
    - assessment of severity
        - blood ketones >6 mmol/L
        - bicarbonate <5 mmol/L
        - venous/arterial pH <7.0
        - hypokalaemia <3.5 mmol/L on admission
        - GCS <12
        - oxygen saturations <92% on air (with normal baseline respiratory function)
        - systolic BP <90 mmHg
        - HR >100 or <60
        - anion gap >16
    - acid-base and electrolyte abnormalities
        - there is usually a severe metabolic acidosis with incomplete respiratory compensation
        - potassium may be normal but the patient will have a whole body potassium deficiency
            - needs to be replaced once <5 mmol/L
        - sodium may be deranged
        - acidaemia rarely requires bicarbonate treatment and will respond to other treatments
    - specific therapy
        - start insulin infusion at 0.1 units/kg/hr
        - aim to lower glucose by 1-2 mmol/L/hr

        - balanced salt solution fluid resuscitation
        - once glucose <15 mmol/L start dextrose 5% at 100ml/hr
            - increase to 10% if needed
        - monitor ketones
        - correct osmolality by 3 mOsm/kg/hr
    - underlying cause
        - treat infection
        - review compliance
        - identify and treat ischaemia
            - ACS
            - CVA
            - PVD
            - mesenteric ischaemia
        - pregnancy
- complications
    - hypoglycaemia
    - hyponatraemia
    - hyperchloraemic acidosis
    - cerebral oedema
    - arrhythmias
    - venous thrombosis
    - infection

EnC3 diabetes mellitus and complications including diabetic foot

- types of diabetes
    - type 1 diabetes mellitus
        - results from the body's failure to produce sufficient insulin
    - type 2 diabetes mellitus
        - results from resistance to insulin
        - often initially has normal or increased levels of circulating insulin
    - gestational diabetes
        - pregnant women who have never had diabetes before but who have high blood glucose levels during pregnancy
        - may precede development of type 2 (or rarely type 1) diabetes
    - maturity onset diabetes of the young (MODY)
        - includes several forms of diabetes with monogenetic defects of beta cell function (impaired insulin secretion)
        - usually autosomal dominant inheritance
        - usually manifests as mild hyperglycaemia at a young age
    - secondary diabetes (rare, 1-2% of cases of diabetes)
        - causes include:
            - pancreatic disease
                - cystic fibrosis
                - chronic pancreatitis
                - pancreatectomy
                - carcinoma of the pancreas
            - endocrine

        - Cushing's syndrome
        - acromegaly
        - thyrotoxicosis
        - phaeochromocytoma
        - glucagonoma
    - drug-induced
        - thiazide diuretics
        - corticosteroids
        - atypical antipsychotics
        - antiretroviral protease inhibitors
    - congenital lipodystrophy
    - acanthosis nigricans
    - genetic
        - Wolfram's syndrome
            - DIDMOAD
                - diabetes insipidus
                - diabetes mellitus
                - optic atrophy
                - deafness
        - Friedreich's ataxia
        - dystrophia myotonica
        - haemochromatosis
        - glycogen storage diseases
- type 1 diabetes
    - about 15% of those with diabetes
    - development is based on a genetic predisposition and an autoimmune process resulting in gradual destruction of beta cells of the pancreas, leading to absolute insulin deficiency
    - there is usually a pre-diabetic phase where autoimmunity has developed and autoantibodies can be detected but there is no clinical insulin dependency
    - possible triggers for the process may include:
        - viruses
        - dietary factors
        - environmental toxins
        - emotional stress
        - physical stress
    - positive family history in around 10%
        - screening in first degree relatives is reasonable although absolute risk is low
    - patients always need insulin therapy and are prone to ketoacidosis
    - the most at-risk population is northern European Caucasians
- type 2 diabetes
    - around 85% of those with diabetes
    - usually >30 years but can occur in children
    - associated with excess body weight and physical inactivity
    - increased prevalence in people of South Asian, African, African-Caribbean, Polynesian, Middle-Eastern and American-Indian ancestry
    - caused by impaired insulin secretion and insulin resistance and has a gradual onset

    - may eventually need insulin treatment
    - risk factors for type 2 diabetes:
        - obesity, especially truncal
        - lack of physical activity
        - ethnicity
        - history of gestational diabetes
        - impaired glucose tolerance
        - impaired fasting glucose
        - drug therapy (e.g. combined use of thiazide diuretic and beta blocker)
        - low fibre, high glycaemic index diet
        - metabolic syndrome
        - polycystic ovary syndrome
        - family history
        - adults who had low birth weight for gestational age
        - statins – risk is likely outweighed by the benefits of reducing cardiovascular risk
- presentation
    - all types
        - polyuria
        - polydipsia
        - lethargy
        - boils
        - pruritus vulvae
        - frequent, recurrent or prolonged infections
    - type 1
        - weight loss
        - dehydration
        - ketonuria
        - hyperventilation
        - presentation tends to be acute with short duration of symptoms
    - type 2
        - presentation tends to be subacute with longer duration of symptoms
    - presentation may be with acute or chronic complications
- diagnosis
    - HbA1c of 48 mmol/mol (6.5%) or more
    - type 2 diabetes is diagnosed with a fasting plasma glucose of 7.0 mmol/L or more if HbA1c is not appropriate (e.g. end stage chronic renal disease)
    - in asymptomatic patients a diagnosis should not be made after one test – at least one further abnormal level is required
    - a second test is prudent in those with symptoms but may not be required
    - severe hyperglycaemia in patients with acute infection, trauma, circulatory or other stress should not be regarded as diagnostic as it may be transient
- primary care management
    - diabetes education
        - structured education and self-management with regular review and reinforcement
    - diet and lifestyle
        - healthy diet

        - weight loss if overweight
        - smoking cessation
        - regular physical exercise
    - maximising glucose control whilst minimising adverse effects such as hypoglycaemia
    - reduction of other risk factors for complications of diabetes
        - including management of hypertension, hyperlipidaemia and consideration of antiplatelet therapy with aspirin
    - monitoring and early intervention for complications of diabetes
        - including cardiovascular disease, foot problems, eye problems, kidney problems, neuropathy
- chronic complications
    - diabetic nephropathy
        - background
            - people with diabetes have increased risk of:
                - renal atherosclerosis
                - urinary tract infections
                - papillary necrosis
                - glomerular lesions
            - nephropathy may be diffuse or nodular
            - the main feature of diabetic nephropathy is proteinuria
                - starts as intermittent microalbuminuria, progresses to constant proteinuria and occasionally nephrotic syndrome
            - clinical features are usually absent until advanced chronic kidney disease develops
            - annual review should occur for all patients 12 years and over
                - urinary albumin creatinine ratio or albumin concentration are measured, using a first morning sample where possible
            - non diabetic causes of renal impairment should also be considered
        - management
            - primary prevention
                - optimal control of blood glucose and blood pressure
                - smoking cessation
            - management of renal impairment
                - good blood glucose control
                - aggressive management of cardiovascular risk factors
                - ACEi titrated to full dose
                    - caution in peripheral vascular disease, known renovascular disease or reduced glomerular filtration rate
                - maintain blood pressure <130/80 mmHg
        - prognosis
            - renal disease accounts for 21% of deaths in patients with type 1 diabetes
            - in type 2 diabetes, when proteinuria and hypertension are present, standardised mortality ratio is increased five fold in men and eight fold in women
    - diabetic retinopathy

- background
    - chronic progressive, potentially sight-threatening disease of the retinal microvasculature associated with prolonged hyperglycaemia and other linked conditions such as hypertension
    - most common eye problem caused by diabetes
        - other problems include:
            - cataracts
                - most commonly earlier formation of age-related cataracts
                - diabetic cataracts manifest as snowflake opacities in a young person with diabetes which may resolve spontaneously or mature
            - rubeosis iridis and glaucoma
                - rubeosis iridis is where severe ischaemia causes neovascularisation to such an extent that vessels grow forward and over the iris
                - can be large individual entities or give the iris a generally red appearance
                - can precipitate acute glaucoma by blocking the trabecular meshwork
            - ocular motor nerve palsies
                - presumably due to damage of the microvascular supply of the cranial nerves
                    - often resolves over a few months if related to diabetic microvasculapathy
                - imaging required to rule out intracranial mass
- pathophysiology
    - not entirely understood
    - microvascular occlusion causes retinal ischaemia leading to arteriovenous shunts and neovascularisation
    - characteristic features include:
        - microaneurysms
            - physical weakening of the capillary walls
            - predisposes them to leakages
        - hard exudates
            - precipitates of lipoproteins/other proteins leaking from retinal blood vessels
        - haemorrhages
            - rupture of weakened capillaries, appearing as small dots, larger blots or ‘flame’ haemorrhages that track along nerve fibre bundles in superficial retinal layers
        - cotton wool spots
            - build-up of axonal debris due to poor axonal metabolism at the margins of ischaemic infarcts
        - neovascularisation

        - an attempt by residual healthy retina to revascularize hypoxic retinal tissue
- classification
    - based on which part of the retina is affected and the degree of pathology on slit-lamp examination and is not necessarily correlated to the degree of visual loss
    - diabetic retinopathy:
        - background (mild) non-proliferative
            - at least one microaneurysm
        - moderate non-proliferative
            - microaneurysms or intraretinal haemorrhages +/- cotton wool spots, venous beading, intraretinal microvascular abnormalities
        - severe to very severe non-proliferative
            - a minimum number of the above features in a minimum number of retinal quadrants
        - non-high-risk proliferative
            - new vessels on the disc or within one disc diameter of it or new vessels elsewhere
        - high-risk proliferative
            - large new vessels on disc or elsewhere or evidence of pre-retinal haemorrhage; there may be accompanying retinal detachment in advanced disease
    - diabetic maculopathy
        - focal or diffuse macular oedema
            - areas of leakage which may be well circumscribed or diffuse
        - ischaemic maculopathy
            - clinical appearance may be relatively normal but visual acuity has dropped and ischaemia seen on fluorescein angiography
        - clinically significant macular oedema
            - there may be thickening of the retina and hard exudates which define CSMO when found within a specific distance of the fovea or above a certain size
- risk factors
    - poor glycaemic control
    - systemic hypertension
    - diabetes duration
    - dyslipidaemia
    - microalbuminuria
    - pregnancy
    - minority ethnic communities within the UK
    - intraocular surgery may increase risk of progression
- history
    - vision may remain normal

- painless gradual reduction of central vision
    - can also be associated with cataract formation
- haemorrhages result in the sudden onset of dark, painless floaters which may resolve over several days
- severe haemorrhage may obscure the vitreous altogether resulting in a painless visual loss
- acute glaucoma preceded by rubeosis iridis will present with acute pain and requires urgent referral

- examination
    - visual acuity
        - acute reduction suggests the need for urgent referral and prognostic caution
    - slit lamp magnification of the fundus or fundal photography
        - requires dilation of the pupil for best view, but the patient will not be able to drive for up to six hours afterwards
        - check red reflex first
            - spots within this suggest vitreous haemorrhage
            - start at the disc and work systematically out along each main arterial branch
                - note little red dots (dot haemorrhages or small aneurysms), irregular notching (venous beading), new vessels (tend to be thinner and more disorganised than pre-existing vessels), well-demarcated creamy/yellow lesions (hard exudates) or paler lesions with less well-defined edges (cotton wool spots)
                - haemorrhages over the macula is an important finding for CSMO
            - end with the macula – patient looks directly at the light
- ophthalmic intervention
    - not always required
    - laser treatment
        - aims to induce regression of new blood vessels and reduce central macular thickening
        - can stop progression but is unlikely to restore vision
    - intravitreal steroids
        - poor evidence base and requires careful consideration of risks and benefits
    - fluocinolone acetonide implant
    - anti-vascular endothelial growth factor treatments
    - surgery
        - vitrectomy
- complications
    - visual loss secondary to:
        - macular oedema

        - macular ischaemia
        - vitreous haemorrhage
        - tractional retinal detachment
    - complications of focal/grid photocoagulation
        - impaired central vision
        - paracentral scotoma
        - choroidal neovascularisation
        - epiretinal membrane formation
        - worsening of macular oedema in a minority
    - complications of panretinal photocoagulation
        - constriction of visual field
        - nocturnal diminution of vision
        - burns affecting the fovea centralis
        - worsening macula oedema
        - serous and/or choroidal detachment
        - ocular pain
        - anterior chamber adverse effects – e.g. burns affecting the cornea or lens
    - complications of intravitreal steroids and triamcinolone
        - cataract formation
        - raised intraocular pressure
- prognosis
    - if left untreated 50% of those with proliferative diabetic retinopathy lose their sight within 2 years and 90% risk losing any useful vision after 10 years

- diabetic neuropathy
    - background
        - common complication of type 1 and type 2 diabetes
        - plays a major role in the development of foot ulcers
        - motor, sensory and autonomic fibres may all be affected
    - risk factors
        - smoking
        - age >40
        - history of periods of poor glycaemic control
        - prevalence increases with increased duration of diabetes
        - people with signs of neuropathy are likely to also have evidence of diabetic nephropathy and retinopathy
        - hypertension
        - coronary heart disease
    - presentation
        - depends on type of neuropathy involved
        - 50% may have no symptoms and are only diagnosed by careful regular clinical examination
        - peripheral sensorimotor (chronic peripheral neuropathy)
            - sensory nerves are affected more than motor
            - touch, pain and temperature sensation and proprioception in lower limbs in a glove and stocking distribution

    - loss of ankle jerks, and later knee jerks
    - hands only affected in severe longstanding neuropathy
    - equal prevalence in types 1 and 2
- acute diffuse painful (acute peripheral neuritis)
    - often abrupt onset and not related to duration of diabetes
    - can resolve completely
    - burning foot pain, often worse at night
    - associated with poor glycaemic control but sometimes initially follows establishing good glycaemic control
    - examination may be normal apart from hyperaesthesia
- acute painful neuropathy of rapid improvement of blood glucose control
    - acute painful neuropathy resulting from rapid improvement of blood glucose control – self-limiting condition that improves symptomatically over time
- autonomic neuropathy
    - more common in females
    - risk factors include hypertension and dyslipidaemia
    - tends to be associated with peripheral neuropathy
    - affects people with type 1 and type 2 diabetes
    - high mortality rate (50% within 3 years) mainly due to CKD but often no obvious cause
    - tight glycaemic control reduces risk
    - may present with:
        - cardiac autonomic neuropathy
            - linked to:
                - resting tachycardia, postural hypotension, orthostatic bradycardia and orthostatic tachycardia
                - exercise intolerance
                - decreased hypoxia-induced respiratory drive
                - loss of baroceptor sensitivity, increased intra-operative or peri-operative cardiovascular lability
                - increased incidence of asymptomatic myocardial ischaemia, myocardial infarction, decreased rate of survival after myocardial infarction
                - congestive heart failure
    - genitourinary
        - impotence, retrograde ejaculation, urinary hesitancy, overflow incontinence
        - often no association with glycaemic control or duration or severity of diabetes
    - gastrointestinal

            - nausea and vomiting
            - abdominal distension
            - dysphagia
            - diarrhoea
        - gustatory sweating, anhidrosis
    - mononeuropathy
        - external pressure or entrapment (e.g. carpal tunnel syndrome)
        - isolated neuropathies of either the cranial or peripheral nerves
        - mononeuropathies of cranial nerves III, IV and VI, intercostal nerves and femoral nerves are common
        - occasionally more than one nerve is involved (mononeuritis multiplex)
    - proximal motor (diabetic amyotrophy)
        - main motor manifestation
        - severe pain and paraesthesia in the upper legs, with weakness and muscle wasting of the thigh and pelvic girdle muscles
        - may be asymmetrical and there may be extensor plantars
        - mainly affects middle aged and elderly patients
        - usually associated with a period of very poor glycaemic control, sometimes with dramatic weight loss
        - pain and weakness gradually reduce once good glycaemic control has returned
- differential
    - toxins
        - e.g. alcohol, occupational, vitamin B6
    - medications
        - e.g. amiodarone
    - hypothyroidism
    - pernicious anaemia
    - malignancies
    - amyloidosis
    - collagen vascular disease, neurosarcoidosis
    - tabes dorsalis
    - AIDS
    - spinal cord disease, cauda equina syndrome
- investigations
    - full assessment of diabetes and blood pressure control
    - assessment of other possible causes (e.g. TFTs, B12)
    - may require nerve conduction studies and electromyography
- management
    - general
        - regular surveillance for signs of neuropathy to allow early intervention
        - tight glycaemic control

        - prevention of foot trauma
    - management of painful neuropathy
        - bed foot cradles for problems at night
        - simple analgesia taken in advance of symptoms
        - contact dressings
        - drug treatments:
            - amitriptyline
            - duloxetine
            - gabapentin
            - pregabalin
            - tramadol only if acute rescue therapy required
            - capsaicin cream
            - avoid opioids
    - management of autonomic neuropathy
        - cardiovascular effects
            - cardio-active drugs such as ACEi, beta blockers, diuretics, digoxin
        - gastroparesis
            - drugs which increase gastric emptying can be tried (e.g. metoclopramide, domperidone, erythromycin)
            - electrical stimulation can help in some patients
        - diabetic nocturnal diarrhoea
            - may be helped by loperamide, codeine, diphenoxylate
        - gustatory sweating
            - topical glycopyrrolate is sometimes helpful
- diabetic foot
    - background
        - motor, sensory and autonomic fibres may be affected
            - sensory symptoms mean that there is no protection against pressure or heat so trauma can initiate the development of a leg ulcer
            - absence of pain contributes to the development of Charcot foot which further impairs the ability to sustain pressure
            - motor fibre abnormalities lead to undue physical stress and the development of further anatomical deformities (arched foot, toe clawing) and contribute to the development of infection
            - when infection complicates a foot ulcer it can be life- or limb-threatening
        - around 10% of people with diabetes will have a foot ulcer at some time in their lives
        - the most common precipitant is accidental trauma, especially from ill-fitting footwear
        - infection can be divided into:
            - superficial and local
            - soft tissue and spreading (cellulitis)

        - osteomyelitis
- risk factors
    - for foot ulceration:
        - peripheral arterial disease
        - peripheral neuropathy
        - previous amputation
        - previous ulceration
        - presence of callus
        - joint deformity
        - problems with vision and/or mobility
        - male sex
    - risk factors for peripheral arterial disease
        - smoking
        - hypertension
        - hypercholesterolaemia
- presentation
    - diabetic foot ulcers
        - usually painless punched-out ulcers in areas of thick callus
        - +/- superadded infection, pus, oedema, erythema, crepitus, malodour
        - neuro-ischaemic ulcers
            - tend to occur on the margins of the foot
        - neuropathic ulcers
            - tend to occur on the plantar surface of the foot
    - neuropathic foot
        - tends to be warm with dry skin, bounding pulses, distended veins, reduced sensation, callus around the ulcer
    - neuro-ischaemic foot
        - tends to be cool and pink with atrophic skin and absent pulses; the foot may be painful and there is little callus
    - Charcot foot
        - neuro-arthropathic process with osteoporosis, fracture, acute inflammation and disorganisation of foot architecture
        - requires immediate referral to a multidisciplinary foot team
        - characterised by bone and joint degeneration which can lead to a devastating deformity
            - usually presents as a hot swollen foot after minor trauma
        - slight trauma triggers fracture of a weakened bone which increases the load on adjacent bones, leading to gross destruction
            - the process is self-limiting but the persisting deformity greatly increases the risk of secondary ulceration
        - plain x-ray may be normal but a bone scan may show a hot spot

        - damage and developing deformity should be limited by immobilising the foot in a cast; realignment arthrodesis of the hindfoot can sometimes prevent amputation
- examination
    - neuropathy
        - using 10g monofilament
    - limb ischaemia
    - ulceration
    - callus
    - infection and/or inflammation
    - deformity
    - gangrene
    - Charcot arthropathy
- management
    - primary care management includes:
        - education, including appropriate footwear and daily foot checks
        - control of glucose, blood pressure and cholesterol
        - smoking cessation and weight control
        - risk assessment
        - mechanical foot interventions to prevent ulceration
        - antibiotics to manage and prevent infection
        - management of peripheral arterial disease, including bypass surgery
        - wound management, including keeping the wound dry and debridement of dead tissue
    - diabetic foot ulcers
        - offloading using casting
        - control of foot infection
        - control of ischaemia
        - wound debridement
        - wound dressings
    - diabetic foot infection
        - soft tissue or bone sample from base of debrided wound for microbiology (or deep swab if this is not possible)
        - consider x-ray
            - x-ray and inflammatory markers may be normal
            - MRI should be considered
        - follow local antibiotic guidelines
            - prolonged treatment for up to six weeks may be required
- prognosis
    - foot ulcers in people with diabetes have a high risk of needing amputation
    - ulcer recurrence rates are high
    - early detection and effective management can reduce complications
    - survival after amputation is poor

EnC4 hyperosmolar hyperglycaemic state

- background
    - deaths are often due to comorbid conditions such as MI
    - higher mortality rate than DKA
    - part of a continuum with DKA, with insulin resistance predominant over insulin deficiency
- pathophysiology
    - triggers
        - infection
        - MI
        - surgery
        - omission of normal medications
    - decreased insulin or resistance leads to decreased glucose utilisation in skeletal muscle, increased fat and muscle breakdown
        - increased hepatic gluconeogenesis
        - increase in glucagon, cortisol, catecholamines
        - increased blood glucose
        - glycosuria and osmotic diuresis
        - just enough insulin to prevent lipolysis and ketone production
- risk factors
    - elderly
    - type 2 diabetes
    - dementia
    - physical impairment limiting access to water
    - renal dysfunction
    - inappropriate diuretic use
    - steroids
    - beta blockers
    - phenytoin
- common and important causes
    - intercurrent or co-existing illness
        - myocardial infarction
        - infection
            - urinary tract
            - pneumonia
            - cellulitis
            - systemic sepsis
            - dental infection/abscess
        - stroke/TIA/intracranial haemorrhage
        - hyperthermia
        - hypothermia
        - intestinal ischaemia/infarction
        - pancreatitis
        - pulmonary embolism
        - AKI or decompensated CKD
        - any cause of acute abdomen
        - hyperthyroidism

        - burns
        - Cushing's syndrome or ACTH-secreting tumour
        - gastrointestinal bleeding
    - medication-induced
        - metformin during intercurrent illness
        - diuretics (especially thiazide or loop)
        - beta-blockers
        - H2 receptor antagonists
        - dialysis/TPN/glucose containing fluids
        - calcium channel blockers
        - chlorpromazine/other antipsychotics (e.g. olanzapine)
        - carbonic anhydrase inhibitors
        - glucocorticoids (e.g. prednisolone, hydrocortisone)
        - phenytoin and other anticonvulsants
        - substance misuse:
            - alcohol
            - cocaine
            - amphetamines
            - MDMA
    - diabetes-related
        - first presentation of diabetes mellitus
            - unsuspected
            - undiagnosed
        - poor diabetic control/non-compliance
            - intentional
            - accidental
            - self-neglect
            - neglect or abuse by carers/family
- history
    - polydipsia
    - polyuria
    - weight loss
    - weakness
    - slow onset
    - progressive dehydration
    - coma
    - symptoms of causes:
        - MI
        - infection
        - diuretics
        - CVA
        - PE
- examination
    - CVS
        - tachycardia
        - decreased skin turgor
        - sunken eyes

        - dry mouth
    - resp
        - tachypnoea
    - CNS
        - drowsy
        - delirium
        - coma
        - focal or generalised seizures
        - visual changes
        - hemiparesis
- investigations
    - very high osmolality
        - >320 mOsm/kg
    - very high glucose
    - normal ketones
    - hyponatraemia (or pseudohyponatraemia as hyperglycaemia draws water out of cells) or hypernatraemia
    - hypokalaemia
    - hypomagnesaemia
    - normal anion gap
    - pH normally >7.3
        - metabolic acidosis not severe
    - renal dysfunction normally present
- diagnostic criteria
    - serum osmolality >320 mOsm/L
    - serum glucose >33 mmol/L
    - profound dehydration (elevated urea: creatinine ratio)
    - no ketoacidosis
- investigations for cause
    - CXR if suspicion of chest infection
    - compliance with medication
    - ECG and troponin for suspicion of MI
    - FBC
    - CRP
    - blood cultures
    - urine if suspicion of UTI
- management
    - goals
        - correct dehydration
            - often 6-9 litres of fluid loss
        - provide insulin
        - replace electrolytes
        - correct metabolic acidosis
    - resuscitation
        - A: may require intubation if coma and not protecting airway
        - B: mechanical ventilation can minimise work of breathing and manage possible metabolic acidosis

        - C: resuscitation with isotonic fluid until the patient has a normal heart rate and BP
    - specific treatment
        - calculate corrected sodium
            - if hypernatraemic, corrected sodium = measured sodium + glucose/3
            - monitor it as sodium changes for glucose
        - calculate water deficit
            - 0.6 x premorbid weight x (1 – 140/corrected sodium)
        - fluid management in first 24 hours
            - fluid replacement with 0.9% sodium chloride
            - only use 0.45% sodium chloride if osmolality not declining despite adequate positive fluid balance
        - replace other electrolytes as required
            - potassium
                - often requires aggressive replacement with 10-20 mmol/hr
                - ensure patient is not anuric
            - magnesium
            - phosphate
            - calcium
        - fluid management in second 24 hours
            - when glucose is less than 15 mmol/L, use 5% dextrose at 100-250ml/hr and saline
            - metabolic acidosis rarely requires specific treatment as usually responds to volume expansion and insulin therapy
            - encourage the patient to drink as soon as possible
        - general
            - insulin at 0.05 units/kg/h
            - do not allow blood glucose to drop by more than 3-5 mmol/L/h
            - once glucose <15 mmol/L and sodium corrected use 10% dextrose
            - thromboprophylaxis – high risk of VTE
            - diagnose and treat cause
            - should be managed in ITU
- complications
    - delirium
    - coma
    - cerebral oedema
        - prevent by resuscitation with isotonic fluid and slow correction of glucose
    - seizures (focal and generalised)
    - severe dehydration and shock
    - renal failure
    - thrombotic complications
        - VTE
        - stroke
        - acute MI
    - intercurrent events
        - sepsis

        - MI
        - aspiration
    - occlusive events
        - focal CNS signs
        - chorea
        - DIC
        - leg ischaemia
        - rhabdomyolysis
    - fluid overload and congestive heart failure
    - metabolic derangement
        - hypokalaemia
        - hypophosphataemia
        - hypomagnesaemia
        - hypoglycaemia
        - hyperchloraemia with non anion gap metabolic acidosis

EnC5 pituitary disorders

- physiology
    - hypothalamus
        - thermoregulation
            - integrates thermoreceptor input
            - controls activity of heat loss and heat gain mechanisms
        - satiety
            - modulated by glucose, CCK, glucagon, leptin
        - water balance
            - osmoreceptors control ADH release from the posterior pituitary
            - angiotensin II stimulates thirst and ADH release
        - circadian rhythms
            - balance between anterior and posterior hypothalamic stimulation controls sleep-wake cycle
        - pituitary control
            - anterior pituitary by hormones secreted into the long portal vein
                - short-loop feedback
                    - negative feedback from the pituitary on the hypothalamus
                - long-loop feedback
                    - negative feedback from a pituitary target gland (e.g. thyroid, adrenals) on the hypothalamus
            - posterior pituitary by neuronal innervation
        - behaviour
            - punishment and reward centres
        - sexual function
    - pituitary (8 hormones from 2 lobes)
        - anterior pituitary
            - ACTH
                - stimulates cortisol release from the zona fasciculata
                - stimulated by CRH, inhibited by cortisol

- TSH
    - stimulates synthesis and release of T3 and T4
    - stimulated by TRH, inhibited by T3
- FSH
    - stimulated by GnRH, inhibited by circulating sex steroids
    - females:
        - stimulates oestrogen synthesis and ovarian follicle development
    - males:
        - stimulates sperm maturation
- LH
    - females:
        - rapid increase stimulates ovulation and corpus luteum development
    - males:
        - stimulates testosterone synthesis
- GH
    - released in a pulsatile fashion
    - stimulated by GHRH
    - typically high with exercise, hypoglycaemia, stress
    - inhibited by somatostatin and IGF-1
    - generally anabolic effects
        - stimulates lipolysis
        - promotes cell growth and development
- prolactin
    - promotes breast development during gestation and lactation after delivery

- posterior pituitary
    - ADH
        - released in response to osmoreceptors in circumventricular organs detecting a change in osmolality
        - effective at:
            - V1 receptors in vascular smooth muscle, causing vasoconstriction
            - V2 receptors in kidney collecting ducts to increase water reabsorption and on endothelium to increase vWF and factor VIII release
            - V3 receptors in the pituitary to stimulate ACTH release
    - oxytocin
        - uterine contraction
        - let-down reflex (stimulates milk release on suckling)
        - psychological bonding

- pituitary tumours
    - types
        - non-functioning adenomas
        - prolactinomas

        - growth-hormone secreting
        - adrenocorticotrophic hormone secreting
        - TSH-secreting
        - LH/FSH-secreting
    - presentation
        - depends on hormone secreted and pattern of growth within the sella turcica
        - local effects from expanding pituitary mass
            - headache
                - classically retro-orbital or bitemporal
                - worse on waking
                - sudden catastrophic headaches from pituitary apoplexy
                - obstruction of CSF from very large tumours causing hydrocephalus and lateral ventricle expansion
            - visual field defects
                - common but often asymptomatic
                - classically bitemporal hemianopia
                    - can be any unilateral or bilateral field defect
            - ocular nerve palsy
                - causing squint
            - extensive extension into hypothalamus
                - disorders of appetite, thirst, temperature regulation and consciousness
        - anterior pituitary hormonal deficiency
            - panhypopituitarism or varying degrees of loss of any of the hormones
                - tends to occur in the order of LH, GH, TSH, ACTH and FSH
            - common presentation in adults:
                - infertility, oligo/amenorrhoea, decreased libido, erectile dysfunction
                - LH/GH deficiency may result in decreased muscle bulk, decreased body hair, central obesity, small soft testes
            - in children:
                - delayed puberty, impairment of growth
            - diabetes insipidus can rarely be a presenting feature
        - hypersecretion of the pituitary hormone:
            - acromegaly
            - hyperprolactinaemia
            - Cushing's disease
            - thyrotoxicosis
    - investigations
        - endocrine studies for hyposecretion/hypersecretion of hormones
        - MRI scan – preferable to CT
    - management
        - may not require management if small, non-functioning and asymptomatic
        - surgery
            - usually trans-sphenoidal
            - rapid deterioration of vision is an indication for immediate surgery

                - post-operative pituitary dysfunction may result in adrenal insufficiency, diabetes insipidus, SIADH, cerebral salt wasting syndrome
                - neurosurgical complications include visual disturbance, CSF leak, subdural haematoma, epistaxis
            - radiotherapy
                - if tumour incompletely resected or remains hypersecretory
            - medication
                - bromocriptine for prolactin-secreting adenomas
                - somatostatin analogues for GH-secreting adenomas
    - prognosis
        - remission in 90% of microadenomas and 50-60% of macroadenomas
- hypopituitarism
    - causes
        - pituitary tumours
            - e.g. adenoma
        - non-pituitary tumours
            - craniopharyngioma
            - meningioma
            - glioma
            - chordoma
            - ependymoma
            - metastasis
        - infiltrative processes
            - sarcoidosis
            - histiocytosis X
            - haemochromatosis
        - infection
            - cerebral abscess
            - meningitis
            - encephalitis
            - tuberculosis
            - syphilis
        - ischaemia/infarction
            - subarachnoid haemorrhage
            - ischaemic stroke
            - Sheehan's syndrome (postpartum haemorrhage with anterior pituitary infarction)
            - pituitary apoplexy (caused by acute infarction of pituitary adenoma)
        - empty sella syndrome
            - radiological diagnosis of absence of normal pituitary within the sella turcica
            - usually benign and asymptomatic but may develop headaches and hypopituitarism
        - iatrogenic
            - irradiation
            - neurosurgery

                - withholding chronic glucocorticoid replacement
            - head injury
                - may have occurred several years before
            - congenital
                - Kallman's syndrome
            - autoimmune
                - lymphocytic hypophysitis
            - pituitary hypoplasia or aplasia
            - genetic causes
            - idiopathic
    - investigations
        - blood glucose
        - U&Es
        - hormonal assays:
            - TFTs,prolactin, gonadotrophins, testosterone, cortisol
            - pituitary function tests
        - cranial MRI
    - presentation
        - varies from asymptomatic to acute pituitary failure
        - ACTH deficiency
            - adrenal insufficiency and Addison's disease
            - chronic
                - fatigue
                - pallor
                - anorexia
                - weight loss
            - acute
                - weakness
                - dizziness
                - nausea
                - vomiting
                - circulatory collapse
                - fever
                - shock
            - children
                - delayed puberty
                - failure to thrive
            - biochemical/physiological
                - hypoglycaemia
                - hypotension
                - anaemia
                - lymphocytosis
                - eosinophilia
                - hyponatraemia
        - TSH deficiency
            - tiredness
            - cold intolerance

- constipation
- hair loss
- dry skin
- hoarseness
- cognitive slowing
- weight gain
- bradycardia
- hypotension
- children:
    - delayed development
    - growth restriction
    - intellectual impairment

- gonadotrophin deficiency
    - women:
        - oligomenorrhoea
        - loss of libido
        - dyspareunia
        - infertility
        - osteoporosis
    - men:
        - loss of libido
        - impaired sexual function
        - mood impairment
        - loss of facial, scrotal and body hair
        - decreased muscle mass
        - osteoporosis
        - anaemia
    - children:
        - delayed puberty
- growth hormone deficiency
    - short stature
    - decreased muscle mass and strength
    - visceral obesity
    - fatigue
    - decreased quality of life
    - impairment of attention and memory
    - dyslipidaemia
    - premature atherosclerosis
    - growth restriction in children
- antidiuretic hormone deficiency
    - polyuria
    - polydipsia
    - decreased urine osmolality
    - hypernatraemia

- management
    - appropriate hormone replacment
- hypopituitary coma

        - usually occurs in a patient known to have hypopituitarism
        - often develops gradually, may occur suddenly due to pituitary apoplexy
        - may be triggered by infection, trauma, surgery, hypothermia, pituitary haemorrhage
        - clinical features include:
            - hormone deficiencies
            - meningism
            - visual field defects
            - ophthalmoplegia
            - reduced consciousness
            - hypotension
            - hypothermia
            - hypoglycaemia
        - management:
            - urgent IV hydrocortisone
            - T3 replacement after steroid treatment
            - pituitary apoplexy requires urgent surgery
- Cushing's syndrome
    - causes
        - exogenous
            - glucocorticoid use (most common cause)
        - endogenous
            - corticotropin-dependent
                - 80% due to pituitary adenomas (Cushing's disease)
                - 20% due to ectopic corticotropin syndrome
                    - can occur with almost any endocrine tumour
                        - usually small cell lung tumour or bronchial carcinoid tumours
            - corticotropin-independent
                - usually due to a unilateral tumour
                    - adrenal adenoma or adrenal carcinoma
            - pseudo-Cushing's syndrome has some or all of the features with hypercortisolism but is not caused by pituitary-adrenal axis problems
                - causes include:
                    - chronic severe anxiety and/or depression
                    - prolonged excess alcohol consumption
                    - obesity
                    - poorly controlled diabetes
                    - HIV
        - presentation
            - truncal obesity, supraclavicular fat pads, buffalo hump, weight gain
            - facial fullness, moon facies, facial plethora
            - proximal muscle wasting and weakness
            - diabetes or impaired glucose tolerance
            - gonadal dysfunction, reduced libido
            - hypertension

                - nephrolithiasis
                - skin: skin atrophy, purple striae, easy bruising, hirsuitism, acne, pigmentation (with ACTH-dependent causes)
                - psychological problems: depression, cognitive dysfunction, emotional lability
                - osteopaenia, osteoporosis
                - oedema
                - irregular menses
                - thirst, polydipsia, polyuria
                - impaired immune function: increased infections, poor wound healing
                - growth restriction in children
        - investigations
            - usually done in secondary care and should be done at the time of intercurrent illness or crisis
            - 24 hour urinary free cortisol
            - 1mg overnight dexamethasone suppression test
            - late night salivary cortisol
        - management
            - tumour resection where possible
            - medical treatment prior to surgery or for unresectable tumours
                - includes metyrapone and ketoconazole
        - complications
            - metabolic syndrome
            - hypertension
            - impaired glucose tolerance and diabetes
            - obesity
            - hyperlipidaemia
            - coagulopathy, thrombophilia
            - osteoporosis
            - perforated viscera
            - impaired immunity
- acute pituitary failure
    - background
        - usually occurs in patients with pre-existing pituitary adenoma
        - evolves within hours or days
        - most cases occur in the fifth decade of life
        - male predominance
    - precipitating factors
        - systemic hypertension
        - major surgery, particularly coronary artery bypass
        - dynamic pituitary function tests
        - anticoagulation therapy
        - coagulopathies
        - oestrogen therapy
        - initiation or withdrawal of dopamine receptor agonist
        - radiation therapy

        - pregnancy
        - head trauma
        - Sheehan's syndrome
            - rare in developed countries
            - results from hypotension caused by massive haemorrhage during or after delivery
            - usually subacute presentation with failure to lactate, breast involution and amenorrhoea
    - history
        - sudden onset
        - headache (95% of patients)
            - often starts as unilateral retro-orbital headache then becomes more generalised
            - thought to be caused by stretching of the dura mater in the sella
        - vomiting (69%)
            - may be caused by intracranial hypertension or meningeal irritation
        - diplopia
        - visual field defects (64%)
            - caused by compression of the optic chiasm
        - ptosis
    - examination
        - may have rapid progression to coma
            - suggestive of stroke, subarachnoid haemorrhage or meningitis
        - bitemporal superior quadrantic field deficit
        - ocular paresis and diplopia
            - impairment of cranial nerves III, IV, VI by cavernous sinus compression
        - Horner's syndrome
        - stroke
        - hypothermia
            - caused by hypothalamic involvement
        - endocrine deficiencies
        - complete ophthalmoplegia
    - assessment
        - formal assessment of visual fields
        - U&Es, LFTs, blood glucose, clotting screen, FBC, random cortisol, prolactin, TFTs, insulin-like growth factor, growth hormone, LH, FSH, testosterone in men, oestradiol in women
        - urgent MRI
            - pituitary CT if MRI contraindicated or not available
    - management
        - resuscitation with correction of fluid and electrolyte abnormalities
        - IV hydrocortisone after baseline endocrine tests in haemodynamically unstable patients
            - 100-200mg bolus then 2-4mg per hour IV infusion
            - indications are haemodynamic instability, altered level of consciousness, reduced visual acuity, severe visual field defects

        - urgent transsphenoidal surgical decompression if remaining clinically and neurologically unstable
    - prognosis
        - difficult to diagnose and treat
        - life-threatening with high mortality
        - complete recovery can be achieved with rapid diagnosis and treatment

EnC6 thyroid emergencies

- thyroid storm
    - background
        - can lead to organ failure and death
        - challenging to diagnose
        - rare – the main challenge is knowing when to consider it
    - when to consider
        - patient with known hyperthyroidism plus any acute illness/deterioration
        - new onset atrial fibrillation and/or dilated cardiomyopathy
        - new onset delirium/psychosis plus abnormal vital signs (fever, tachycardia)
        - hyperthermia (>40°)
        - septic-appearing patient without any focus of infection
    - precipitants
        - general stressors
            - infection (most common)
            - surgery or trauma (especially neck trauma such as strangulation)
            - pulmonary embolism, myocardial infarction, stroke
            - labour, pre-eclampsia
            - diabetic ketoacidosis, hypoglycaemia
        - thyroid related
            - thyroid surgery, radio-iodine therapy
            - non-compliance with anti-thyroid medications
            - overdose of thyroid hormone
            - acute iodine load from contrast dye or amiodarone
            - checkpoint inhibitors, Sorafenib
        - no identifiable precipitant
            - 30% of patients
    - signs and symptoms
        - usually presents as a mimic:
            - neuro-type mimics
                - psychosis mimic
                - meningitis mimic
                - hyperthermia mimic
            - cardiac-type mimic
                - sepsis mimic
                    - fever
                    - vasodilatory shock
                - heart failure
                    - can drop ejection fraction
                - AF with intractably elevated heart rate

        - cardiac (often predominant feature)
            - tachycardia, atrial fibrillation
            - high output distributive heart failure
            - systolic heart failure can occur
        - neurological
            - delirium
            - agitation
            - psychosis
            - stupor/coma
        - hyperthermia (nearly universal)
            - may reach 40-41°
            - usually has associated diaphoresis
        - gastrointestinal
            - diarrhoea, nausea, vomiting
            - abdominal pain
            - jaundice, hepatic failure
        - other features suggesting hyperthyroidism
            - tremor
            - goitre, scar from partial thyroidectomy
            - exophthalmos
    - investigations
        - diagnosis is clinical – lab results are not worse than in general hyperthyroidism
        - key results:
            - low TSH
            - high free T4 and free T3
        - may also have:
            - hyperglycaemia
            - hypercalcaemia
            - low or high WCC
            - abnormal LFTs
    - diagnostic criteria (may be helpful but clinical judgment still important) – Burch criteria
        - temperature
            - 37.2-37.7 = 5
            - 37.8-38.2 = 10
            - 38.3-38.8 = 15
            - 38.9-39.4 -= 20
            - 39.5-29.9 = 25
            - 40+ = 30
        - tachycardia
            - 99-109 = 5
            - 110-119 = 10
            - 120-129 = 15
            - 130-139 = 20
            - >140 = 25

        - CNS effects
            - mild (e.g. agitation) = 10
            - moderate (e.g. delirium, psychosis) = 20
            - severe (e.g. seizure, coma) = 30
        - gastrointestinal
            - diarrhoea, nausea and vomiting, abdominal pain = 10
            - unexplained jaundice = 20
        - heart failure
            - mild (e.g. oedema) = 5
            - moderate (e.g. rales) = 10
            - severe (e.g. pulmonary oedema) = 15
        - atrial fibrillation
            - present = 10
        - precipitant history
            - present = 10
        - interpretation:
            - 45 or more = highly suggestive of thyroid storm
            - 25-44 = supports diagnosis of thyroid storm or impending storm
            - <25 = thyroid storm unlikely
    - management
        - endocrine
            - steroids
                - blocks release of T4 from the thyroid and peripheral activation of T4 into T3
                - 300mg IV hydrocortisone loading dose
                    - maintenance 100mg TDS
                    - can be rapidly tapered after clinical improvement
            - thionamides
                - block thyroid hormone synthesis
                    - may be ineffective in cases due to thyroiditis
                - methimazole
                    - safer and less hepatotoxic
                    - 40mg loading dose then 20mg 4 hourly
                - propylthiouracil
                    - theoretically more effective
                    - more hepatotoxic
                    - may be preferred in more fulminant and definite cases of thyroid storm and in pregnancy
                    - 200mg dose
            - iodine
                - immediately suppresses thyroid hormone release vis the Wolff-Chiakoff effect
                - must be given at least an hour after thionamide to prevent increasing thyroid hormone synthesis
                - may be continued for up to 10 days – the suppressive effect will eventually wear off
                - lithium may be used instead for patients allergic to iodine

- cholestyramine
    - binds thyroid hormone in the gut and prevents enterohepatic reabsorption
    - effective even in patients who have not taken exogenous thyroid hormone (Graves' disease)
    - extremely safe, and available over the counter for treatment of diarrhoea
    - 4g PO 6 hourly, continued until patient is fully improved
- cardiovascular
    - volume replacement
        - based on echo, lung sonography and history (e.g. poor oral intake, fever, diarrhoea)
    - vasopressors may be required to maintain BP (consider phenylephrine if patient already very tachycardic)
    - magnesium for patients with AF and rapid ventricular rate; hyperthyroidism can also cause hypomagnesaemia
    - beta blockers
        - avoid if possible – may exacerbate shock, particularly in patients with heart failure
            - moderate degree of compensatory tachycardia may be required to maintain adequate perfusion
        - patient must have full haemodynamic assessment including echo and lung sonography before starting
        - esmolol infusion is probably the safest and can be titrated upwards if tolerated
        - propranolol blocks peripheral activation of T4 to T3
        - starting dose is 20-40mg PO QDS, can be uptitrated
- hyperthermia
    - occurs due to increased heat generation by tissues rather than a change in the hypothalamic set point
    - hyperthermia is harmful due to increased cardiac workload and organ damage (e.g. rhabdomyolysis, delirium)
        - induction of shivering is also potentially dangerous – also increases cardiac workload
        - consider paracetamol
        - cooling blankets can be used for high fever if tolerated without shivering
        - salicylates and NSAIDs should be avoided (may increase free thyroid hormone levels)
- agitation
    - may worsen hyperthermia or impede management
    - olanzapine PO, IM or IV can be used
- refractory thyroid storm
    - failure to respond to optimal medical management
        - clinical improvement should be seen within 24-48 hours
    - treatment options include plasmapheresis or thyroidectomy

- decompensated hypothyroidism ('myxoedema coma')

- background
  - generally caused by a background of severe hypothyroidism plus a triggering event
  - may be described as hypothyroidism causing organ failure (with the brain being the first organ to fail usually – delirium)
  - patients are most often female and elderly
  - the majority of cases occur during winter
- triggers
  - nonadherence to thyroid supplementation
  - medications
    - sedatives
    - opioids
    - diuretics
    - beta-blockers
    - amiodarone
    - antipsychotics
    - lithium
    - checkpoint inhibitors
  - infection
  - surgery, trauma, burns
  - MI or heart failure
  - CVA
  - GI bleed
  - some iodinated contrast dyes may cause transient thyroid suppression
  - physical stress
    - cold exposure
- clinical features
  - altered mental status
    - usually hypoactive delirium rather than frank coma
    - rarely, an activated form known as 'myxoedema madness'
  - one of two cardinal features:
    - hypothermia (may be severe)
    - bradycardia
  - other features
    - features of the precipitating events
    - hypoglycaemia
    - hyponatraemia
    - hypoventilation
    - reduced bladder and bowel motility
  - clues to diagnosis of hypothyroidism
    - thyroidectomy scar or goitre, history of prior thyroid disease of any kind
    - myxoedema
      - non-pitting oedema of hands, ankles, face
      - hoarseness, macroglossia
    - hair loss, loss of outer third of eyebrows
    - cold intolerance

        - cardiovascular
            - bradycardia
            - pericardial effusion and pleural effusions
                - can impair diastolic filling of the heart
            - cardiogenic shock (late finding)
                - hypothyroid patients often have diastolic hypertension – it may be a poor sign when the diastolic starts falling
                - shock may result from a combination of bradycardia, pericardial effusion, impaired contractility and/or peripheral vasoconstriction
                    - shock may be vasopressor resistant until thyroid replacement is administered
            - torsades de pointes can occur
        - neurological
            - delirium or coma
            - seizures can occur, including status epilepticus (potentially exacerbated by hyponatraemia)
        - pulmonary
            - hypoventilation due to central reduction in respiratory drive and muscular weakness
            - pleural effusions can promote hypoxaemia
            - oedema of the tongue can complicate intubation
        - haematological
            - acquired von Willebrand syndrome can occur
                - factors V, VII, VIII, IX, X may also be reduced
                - may be treated with desmopressin
            - anaemia is common
        - renal
            - hyponatraemia usually results from excessive levels of anti-diuretic hormone (ADH)
            - AKI may result from hypoperfusion and possible rhabdomyolysis
            - reduced bladder motility may cause urinary retention
        - gastrointestinal
            - ileus may cause nausea and vomiting
            - hypothyroidism may lead to megacolon
            - ascites can occur (not common)
            - GI bleeding as a result of coagulopathy
    - investigations
        - bloods
            - TSH, free T4, random cortisol
                - TSH will be markedly elevated (except possibly with central hypothyroidism due to pituitary insufficiency)
                - free T4 will be decreased
                - diagnosis of decompensated hypothyroidism is clinical, labs tests cannot differentiate
            - elevated CK and leucopaenia can also occur
        - ECG

- bradycardia (most common), conduction blocks
- QTc may be prolonged and may lead to torsades des pointes
- low voltage complexes

- evaluation for triggers
  - infection screen
  - CT head
  - LP

- differential
  - adrenal crisis
  - septic shock
  - meningitis
  - hypothermia of other aetiology
  - CVA
  - drug intoxication
    - e.g. carbon monoxide, clonidine, beta-blocker, calcium-channel blocker
- management
  - aggressive investigation and treatment of trigger
    - consider empirical antibiotics
    - decompensated hypothyroidism can mask features of sepsis
  - hypothermia
    - supportive care
    - may be risk of peripheral vasodilation and circulatory collapse with warming, but not much evidence to support it
    - warm humidified oxygen via high flow nasal cannulae is another option
  - hypoglycaemia
    - may require IV dextrose
    - should improve with steroid administration
  - hyponatraemia
    - may contribute to delirium and seizures
    - manage as usual
      - repeat U&Es, calcium, magnesium, phosphate, glucose; serum osmolality; TSH and cortisol; urine osmolality and sodium
      - treat only if acute and symptomatic:
        - seizure
        - confusion
        - headache, nausea, vomiting
        - dizziness, gait instability, tremor, multifocal myoclonus
      - treatment options:
        - hypertonic sodium bicarbonate (8.4%/ 1mEq/ml)
          - 100ml will raise serum sodium by around 3mM
          - infuse slowly over 10-20 minutes
          - avoid in metabolic alkalosis

        - hypertonic saline (3%)
            - 2ml/kg (around 150ml)
            - safe to administer through a peripheral line
        - repeat electrolytes after correction (aim is 3-5mM increase – should cause clinical improvement)
            - if symptoms have not resolved after 6mM increase, look for another cause of symptoms
    - mild hyponatraemia resolves with thyroid supplementation
- respiratory
    - may require intubation and ventilation due to coma
    - macroglossia may make intubation difficult
- cardiovascular
    - tamponade may require drainage
        - avoid pericardiocentesis if possible as patients tend to bleed and effusions improve with thyroid hormone replacement
    - titrated fluid for volume replacement
    - shock may be pressor-refractory until thyroid treated
- haematological
    - consider desmopressin 0.3 microgram/kg IV for haemorrhage
- hormones
    - hydrocortisone
        - 100mg IV then TDS
        - should be given before thyroid hormone replacement
            - can precipitate adrenal crisis otherwise (adrenal insufficiency often associated with hypothyroidism due to pituitary disease or multifocal autoimmune disorder)
        - can be weaned off rapidly once stable and improving
    - levothyroxine (T4)
        - loading dose of 200-400 microgram IV
            - lower end of range for people who are elderly, have low BMI, coronary artery disease, arrhythmia or liothyronine treatment
        - can be given empirically if bloods delayed
            - inactive form of hormone so no immediate effect
            - normal amount of circulating hormone is around 1000 microgram, so loading dose will not have major effect even if not hypothyroid
        - maintenance dose 1.2 microgram/kg/day IV (or 100 microgram)
        - oral replacement should not be used as GI absorption may be erratic
    - liothyronine (T3)
        - activated form of thyroid hormone
        - can be used in combination with T4 to start recovery

- should not be given empirically – need to wait for test results
  - can precipitate arrhythmias
  - does not have to be given and should be reserved for sicker patients
- may also be given orally

hyponatraemia

- severity
  - <135 mmol/L
    - mild: 125-134
    - moderate: 120-124
    - severe: <120
- types and causes
  - hypo-osmolar/hypotonic
    - hypovolaemia
    - euvolaemic
    - hypervolaemic
    - causes
      - solute depletion or solute dilution
      - hypovolaemic
        - loss of water and sodium from the extracellular fluid
        - leads to increased ADH secretion
        - leads to decreased free water excretion and water retention
        - leads to hyponatraemia
        - renal loss
          - diuretics
          - osmotic diuresis (glucose, mannitol)
          - bicarbonaturia (renal tubular acidosis)
          - salt wasting nephropathy
          - mineralocorticoid deficiency
          - ketones
        - non-renal loss
          - upper GI: vomiting
          - middle GI: pancreatitis, bowel obstruction
          - lower GI: diarrhoea, bowel preparation
          - other losses: sweat, bleeding
        - differentiation
          - urinary sodium < 10 mmol/L – extra-renal (kidney appropriately attempts to conserve sodium)
          - urinary sodium >20 mmol/L – renal (inappropriate response if clinically hypovolaemic)
      - euvolaemic
        - causes:

- SIADH (most common)
    - causes include:
        - malignancy
        - ADH secretion (ectopic)
        - drugs (e.g. SSRIs, ecstasy)
        - CNS disease
        - hormone deficiency (hypothyroidism, adrenal insufficiency)
        - others
- psychogenic polydipsia
- hypotonic IVF therapy
- adrenal insufficiency
- hypothyroidism

- any disease state causing hypo-osmolality can present with a normal state of hydration
- clinical examination of volume status is not sensitive
- a normal or low urea and elevated urinary sodium is more sensitive

- hypervolaemic
    - increase in total body sodium and water, however total body water is out of proportion to sodium
    - clinically evident oedema or ascites
    - causes include:
        - congestive heart failure
        - cirrhosis
        - nephrotic syndrome
        - hypothyroidism
        - pregnancy
        - TURP/hysteroscopy syndrome

- iso-osmolar/isotonic
    - known as 'pseudohyponatraemia'
    - plasma osmolality can be measured directly in the lab or calculated: (2x Na^+) + glucose + urea
    - the nonaqueous components of plasma such as lipids and proteins do not normally effect tonicity but where there is marked hyperproteinaemia or hyperlipidaemia the ratio is artifactually changed, decreasing the apparent concentration of sodium in serum
- hypertonic
    - translocational hyponatraemia
    - osmotically active particles in plasma cause movement of water from intracellular fluid to extracellular fluid, decreasing serum sodium even though serum osmolality remains elevated
    - causative agents include:
        - glucose
        - mannitol
        - sorbitol

        - radiocontrast
    - for each 1 mmol rise in blood glucose, serum sodium reduces by 0.3 mmol/L
    - advanced renal disease
        - important cause of normal or high serum osmolality
        - hyponatraemia is caused by the inability to excrete water – this lowers osmolality but high urea acts to normalise osmolality or make it high
        - urea is an ineffective osmole as it freely crosses cell membranes so does not cause water to move out of cells
        - serum osmolality need to be corrected for the effect of urea
            - corrected osmolality is measured osmolality minus urea
- history
    - speed of onset more important than level
    - most patients are not symptomatic until sodium <125 mmol/L
    - fluid intake/output
    - nausea
    - vomiting
    - neuropsychiatric symptoms
    - muscular weakness
    - headache
    - lethargy
    - psychosis
    - raised ICP
    - seizures
    - coma
    - medications
    - co-morbidities (e.g. adrenal disease, liver disease)
- examination
    - cause
    - volume status
    - neurological signs
        - increased ICP, lateralising signs
- investigations
    - U&Es
    - glucose
    - plasma osmolality
    - plasma proteins and lipids
    - urinary sodium
- management
    - general
        - treat cause
        - ideally correct slowly to avoid central pontine myelinolysis
        - quantify duration, severity and type
        - treatment depends on cause
            - options include:

                - fluid restriction (<800ml/day)
                - diuretics
                - isotonic saline in true volume depletion
                - oral sodium tablets
                - hypertonic saline (3%)
                    - rapid reversal: 100ml every 10 minutes until seizures stop (raised sodium by 2-3mmol/L)
                    - less rapid: 1ml/kg/hr lean body weight
            - desmopressin or dextrose can be used if inadvertent rapid correction takes place
        - acute (<48h) and symptomatic
            - seizing and coma
            - raise by 1-5 mmol/L/h until symptoms resolve or sodium 125-130 mmol/L
            - hypertonic saline (3%) 1-2ml/kg/h
            - furosemide 20mg IV
        - chronic symptomatic (>48h or unknown duration)
            - calculate sodium deficit
            - correct sodium by 10 mmol/L/day
            - furosemide 20mg IV
            - normal saline
            - fluid restriction
            - repeat sodium every 2 hours
            - high risk patients are young premenopausal women
        - asymptomatic from SIADH
            - fluid restrict
            - furosemide 20-40mg/day
            - sodium chloride tablets 3-18g/day
            - urea 30g/day
            - demclocycline 200-1200 mg/day
    - normal ranges
        - serum osmolality
            - 272-295 mOsm/kg water
            - panic values 240 and 320
        - urine osmolality
            - 50-1400 mOsm/kg water (average 500-800)
            - after an overnight fast urine osmolality should be 3 times the plasma osmolality
        - urine sodium
            - -15-250 mmol/L
    - calculations
        - sodium deficit
            - total body watert x [sodium desired – sodium measured]
        - rate of infusion (ml/h)
            - sodium requirement (mmol) x 1000/ infusate sodium (mmol/L) x time (hours)
    - total body water

        - children = 0.6 x weight
        - women = 0.5 x weight
        - men = 0.6 x weight
        - elderly women = 0.45 x weight
        - elderly men = 0.5 x weight
    - sodium content of fluids (infusate sodium in mmol/L)
        - 5% sodium chloride = 855
        - 3% sodium chloride = 513
        - 0.9% sodium chloride = 154
        - Hartmann's = 130
        - 0.45% sodium chloride = 77
        - 0.2% sodium chloride = 34
        - 5% dextrose = 0

ENVIRONMENTAL EMERGENCIES

EnvC1 heat stroke and heat exhaustion

- hyperthermia causes
    - exogenous
        - environmental – high temperatures, humidity
    - endogenous
        - prolonged muscular activity – seizures, marathons, excessive dancing after recreational drug
        - drugs – ecstasy, cocaine, amphetamines etc.
        - neuroleptic malignant syndrome – drug reaction to antipsychotics
        - malignant hyperthermia – autosomal dominant condition related to use of suxamethonium or inhaled anaesthetic agents
- severity
    - heat cramps
        - core temperature 37-39°
        - usually during exercise when sweat losses replaced by hypotonic fluids
        - sodium levels decreased, muscle cramps develop
        - manage with oral rehydration fluids
    - heat exhaustion
        - core temperature <40°
        - sodium and water depletion
        - symptoms: weakness, fatigue, headache, dizziness, nausea, vomiting, syncope
        - homeostatic mechanisms function but are overwhelmed
        - management:
            - remove from heat
            - oral rehydration fluids for mild cases
            - IV rehydration fluids for more severe cases
    - heat stroke
        - core temperature >40.6°
        - early symptoms similar to heat exhaustion

- progresses to confusion, seizures, coma, hypotension, arrhythmia
- non-exertional heat stroke can occur in the elderly in hot weather and presentation may be less obvious
- all thermoregulatory control lost, multi-organ damage occurs
- complications:
    - CNS – cerebral oedema, petechial hameorrhages
    - renal – AKI secondary to hypovolaemia and rhabdomyloysis
    - liver – raised liver enzymes and jaundice after 24 hours
    - haematological – thrombocytopaenia and DIC
    - metabolic – hyper/hypokalaemia, metabolic acidosis, hypoglycaemia
    - muscle – rhabdomyolysis
- investigations:
    - glucose
    - biochem screen including calcium, magnesium, phosphate, LFTs
    - FBC
    - coag, fibrinogen, d-dimer
    - CK
- management:
    - cooling (aim for rapid reduction to 39°)
        - remove from hot environment
        - remove clothing
        - evaporative cooling: spray with tepid water, blow air over with fans (reduction of around 0.1°/minute)
        - ice packs to neck, axillae and groins
            - consider placing patient in body bag filled with ice
        - advanced cooling techniques – cold IV fluids (1 litre of chilled crystalloid reduced temperature by around 1 degree), intravascular cooling catheters, surface cooling devices, extracorporeal circuits
    - monitor for and treat hypoglycaemia
    - cautious IV fluids
        - risk of cerebral and pulmonary oedema
        - cautious titration of 0.9% sodium chloride if BP remains low as temperature falls (with CVP line and catheter)
        - more rapid if rhabdomyolysis present
    - no evidence for use of antipyretics
    - consider high flow dry air via nasal prongs/nasal CPAP for evaporative cooling (not evidence-based)
    - consider dantrolene if hyperthermia secondary to neuroleptic malignant syndrome or malignant hyperthermia
    - sedation
        - may be needed for agitation/to reduce muscle exertion (never use physical restraints as will increase heat generation through struggling)
        - consider benzodiazepines, ketamine, propofol, opioids, dexmedetomidine
    - shivering

        - control during initial cooling as impairs temperature management
        - non-depolarising muscle relaxant for intubated patients
        - for non-intubated patients consider: IV benzodiazepines, fentanyl, pain dose ketamine infusion (0.15-0.3 mg/kg/hr), IV magnesium, IV ondansetron
    - intubation
        - indications include:
            - unclear cause of hyperthermia and intubation needed for diagnostic tests such as MRI/LP
            - severe rigidity
            - status epilepticus
            - worsening respiratory failure
            - refractory agitations
        - avoid succinylcholine (can confuse diagnosis with possibility of malignant hyperthermia and may worsen hyperkaemia)
    - temperature goals
        - measure by oesophageal probe if intubated, or bladder/rectal probe
        - drop to 38° as fast as possible
        - stop active cooling when temperature <38°
        - continue monitoring for at least 6 hours

EnvC2 drug-related hyperthermias

- serotonin syndrome
    - reaction due to excess serotonergic activity in the CNS and at peripheral serotonin receptors
    - more likely if exposed to two or more drugs that increase serotonin effect at synapses
    - causes:
        - overdose of SSRI
        - concomitant use of SSRI and MAOI (or insufficient time between stopping one and starting another)
        - concomitant use of SSRI and TCA
        - concomitant use of SSRI and tramadol, SNRI, triptan, linezolid, St John's wort
        - use of stimulant drugs of abuse (e.g. ecstasy, amphetamine, cocaine) especially alongside SSRI
    - presentation:
        - may be insidious over minutes to hours
        - triad of altered mental status, neuromuscular hyperactivity and autonomic instability
            - altered mental status (40%)
                - agitation
                - confusion
                - delirium
                - hallucinations

        - drowsiness
        - coma
        - seizures
    - neuromuscular hyperactivity (50%)
        - tremor
        - shivering
        - teeth grinding
        - myoclonus
        - hyperreflexia
    - autonomic instability (50%)
        - tachycardia
        - hyperthermia
        - hyper- or hypotension
        - flushing
        - diarrhoea and vomiting
- investigations:
    - U&E
    - CK
    - ABG
    - coag
    - urine for myoglobin
    - glucose
    - ECG
- management:
    - stop all potential causative drugs
    - supplemental oxygen for hypoxia +/- intubation and ventilation
    - IV fluids for hydration and treatment of hypotension
        - monitor urine output
    - IV benzodiazepines for frequent or prolonged seizures (consider phenobarbital as second line)
    - external cooling for mild hyperthermia, rapid cooling for temperature >39° (ice baths, sedation) – consider dantrolene
    - IV fluids +/- urinary alkalinisation for rhabdomyolysis
        - haemofiltration if needed
    - cyproheptadine (on NPIS advice)

- neuroleptic malignant syndrome
    - complication of neuroleptic drugs (idiosyncratic reaction after prolonged exposure to neuroleptics or withdrawal of dopamine receptor agonist)
        - risk factors:
            - phenothiazines (chlorpromazine, promethazine)
            - butyrophenones (droperidol, haloperidol)
            - thioxanthenes (chlorpothixene)
            - benzamides (sulpiride)
            - clozapine
            - risperidone
            - abrupt stopping of neuroleptic or PD drugs
            - alcoholics

        - exhaustion
        - dehydration
        - malnutrition
    - possibly due to alteration of central neuroregulatory mechanisms causing impaired heat dissipation, or abnormal reaction of skeletal muscle
    - presentation:
        - over 24-72h (usually develops over days or weeks)
        - hyperthermia
        - rigidity
        - rhabdomyolysis
        - encephalopathy/disturbed consciousness
        - respiratory – decreased chest wall compliance, tachypnoea, pulmonary infection
        - neurological – dyskinesia, dysarthria, parkinsonism, agitation, stupor, coma, seizures, chorea, trismus
        - renal – renal failure
        - CVS – tachycardia, high BP, autonomic dysfunction
        - haematological – high WCC
        - hepatic – raised LFTs
    - differential:
        - serotonin syndrome
        - malignant hyperthermia
        - CNS infection
        - vasculitis
        - heat stroke
        - drug toxicity (MAOI, lithium, cholinergic)
    - investigations:
        - CK
        - urine myoglobin
        - U&Es/LFTs
        - FBC
        - coag (risk of DIC)
    - management:
        - discontinue antipsychotic drug
        - cooled IV fluids
            - correct for insensible losses, reduce hyperthermia
        - other cooling techniques as needed
        - if remains hyperpyrexic with muscle rigidity – dantrolene
        - NPIS may advise bromocriptine (can cause recurrence of psychotic symptoms)
        - sedation, paralysis and ventilation if indicated
        - consider urinary alkalinisation for rhabdomyolysis (1.5 litres of 1.26% sodium bicarbonate or 225ml 8.4%) – aim for urine pH >7.5
            - may require haemofiltration
    - complications:
        - death (12-20%)
        - renal failure
        - rhabdomyolysis

        - DIC
        - thrombocytopaenia
        - electrolyte disturbance
- malignant hyperthermia
    - rare autosomal dominant condition, disease of skeletal muscle
        - mutation in gene coding for ryanodine receptor on chromosome 19
        - excess calcium release during muscle contraction increases muscle metabolism and heat production
        - prolonged and intensified actin and myosin interaction
        - enhanced aerobic metabolism leading to lactic acidosis, accumulation of intra-mitochondrial calcium, deconjugation of oxidative phosphorylation and cytolysis
    - triggers:
        - all volatile agents except nitrous oxide
        - suxamethonium
        - patients may have tolerated the same triggers previously
    - presentation:
        - intra-operative and 4 hours post
        - increased ETCO2
        - tachycardia
        - tachypnoea
        - masseter spasm
            - impedes intubation
            - lasting for 2 minutes
            - not improved with repeat doses of suxamethonium or non-depolarising relaxants
            - do not proceed to use volatile agents after suxamethonium if masseter spasm occurs
        - muscle rigidity
        - temperature increase
        - tachyarrhythmia
        - difficulty ventilating
        - hypertension
        - sweating
        - DIC
        - hyperkalaemia
        - cardiac arrest
    - management:
        - call for help
        - maintain anaesthesia with hypnotics and opioids
        - hyperventilate
        - cooling
        - maintain urine output
        - inotropes as needed
        - dantrolene
    - prognosis:
        - mortality without dantrolene 70%
        - mortality with dantrolene 5%

EnvC3 hypothermia and frostbite

- definition
    - mild – 32-35°
    - moderate 30-32°
    - severe <30°
- Swiss staging system
    - I – clearly conscious and shivering
    - II – impaired consciousness without shivering
    - III – unconscious
    - IV – not breathing
    - V – death due to irreversible hypothermia
- requires low reading thermometer for core temperature
- rapid (e.g. sudden immersion in cold water) or gradual (e.g. immobility, impaired consciousness secondary to alcohol, drugs, illness)
- elderly and very young more susceptible due to impaired thermoregulation
- in cardiac arrest:
    - may be the cause or be secondary to environmental cooling after death
    - death should not be confirmed until patient has been rewarmed or attempts to rewarm have failed
    - modifications to ALS algorithm:
        - palpate a major artery and look for signs of life for up to 1 minute before concluding absent cardiac output
            - use echo if possible to confirm presence or absence of cardiac output
            - if there is doubt, begin CPR
        - heart may be unresponsive to cardio-active drugs and drug metabolism is slower
            - can lead to toxic concentrations
            - withhold cardio-active drugs until core temperature >30°
            - double interval between doses between 30° and 35°
        - if VF/VT detected, defibrillate
            - if persisting beyond 3 shocks, postpone further shocks until >30°
        - interventions such as central line and intubation should be performed by expert to avoid excessive movement and risk of precipitating VF
        - rewarm to 32-34° - therapeutic hypothermia may be beneficial post arrest
        - large volumes of IV fluids may be required as patients rewarm and vasodilate
- effects:
    - CVS – bradycardia, vasoconstriction, wide QRS, prolonged PR and QT, J waves, risk of VF, increased blood viscosity and myocardial work
    - respiratory - decreased CO2, increased dead space, diaphragmatic fatigue, metabolic acidosis, pulmonary hypertension
    - GI – decreased hepatic metabolism and blood flow, decreased splanchnic circulation
    - metabolic – decreased basal metabolic rate, shivering, left shift of oxygen/Hb dissociation curve, hyperglycaemia, decreased drug metabolism
    - CNS – neuroprotection, fixed dilated pupils at <30°

    - haematological – increased bleeding time, PT and APTT, VTE risk, decreased platelet and WCC
    - renal – decreased GFR, cold induced diuresis
- hypothermia features:
    - ataxia
    - dysarthria
    - falling conscious level
    - coma
    - hypotension
    - arrhythmia
        - typically sinus bradycardia becomes atrial fibrillation, then VF and asystole
- investigations:
    - U&Es, amylase (may precipitate pancreatitis)
    - FBC
    - coag
    - toxicology screen
    - glucose
    - ABG
    - ECG
        - prolonged PQRST complex, J waves (delayed repolarisation), arrhythmia
    - CXR
        - aspiration, pneumonia, pulmonary oedema
    - CT head
        - if head injury/CVA suspected
- management:
    - move gently if <32° - risk of VF
    - rewarm at rate of 0.5-2°/hour
        - rapid rewarming may precipitate pulmonary and cerebral oedema, particularly in the elderly
    - rewarming techniques
        - active
            - remove cold or wet clothing
            - dry patient
            - cover with blankets/hot air blanket
            - chemical heat pads
            - warm room (overhead heaters if available)
        - passive
            - warmed humidified oxygen
            - warmed IV fluids
            - gastric, peritoneal, pleural or bladder lavage with warmed fluids
            - cardiopulmonary bypass
    - cardiac monitoring
        - most arrhythmias other than VF revert as warming occurs
    - regular blood glucose monitoring and correction
    - IV fluids if BP drops
        - 300-500ml warmed 0.9% sodium chloride
        - risk of pulmonary oedema

        - consider CVP line and urinary catheter
    - rewarming rates for different methods:
        - shivering 1.5°/hr
        - warming blanket 2°/hr
        - warm O2 1°/hr with mask, 1.5°/hr ET tube
        - IV fluids do not add but do not take away
        - peritoneal lavage 3°/hr
        - thoracic lavage with chest tubes 3-6°/hr
        - cardiac bypass 9-18°/hr
- frostbite
    - extensive soft tissue damage associated with exposure to temperatures below freezing
        - risk is low at >-10°
        - risk is high at <-25°
        - risk increased with duration of exposure, windchill, altitude, contact with conductive materials (water, ice, metal)
        - increased risk with alcohol abuse, mental illness, peripheral vascular disease, peripheral neuropathy, malnutrition, chronic illness, tobacco use, African descent
        - more common in males (10:1)
    - 4 phases
        - phase I – cooling and freezing
            - vasoconstriction/vasospasm followed by transient arteriovenous shunting, cycles of vasodilatation/vasoconstriction every 10 minutes
            - cycles cease with persistent cold, temperature drops to freezing point of tissue (<2°)
            - ice crystals – extracellular cause sludging/stasis and intracellular dehydration, intracellular destroy cell membranes
            - limb temperature falls to ambient temperature when crystallisation complete
        - phase II – rewarming
            - reverses freezing process, limb absorbs heat and ice crystals melt
            - intracellular swelling
            - endothelial cells of capillaries become permeable, with fluid extravasation leading to blisters/oedema
            - important to prevent refreezing
        - phase III – progressive tissue injury
            - inflammation, stasis/thrombosis, tissue necrosis
            - diminished prostaglandin E2 (vasodilator, antiplatelet)
            - elevated prostaglandin F2a and thromboxane B2 (vasoconstrictors, platelet-aggregating)
        - phase IV – resolution
            - complete healing with no symptoms
            - healing with sequelae
            - early tissue necrosis/gangrene
    - associated conditions:
        - frostnip

                - mildest cold exposure injury
                - superficial layers of skin only (blanching, numbness), no dermis damage
            - chilblain (pernio)
                - in cold, non-freezing temperatures in dry conditions
                - burning sensation with pruritus, swelling, erythema
                - may have blisters, ulceration
                - resolves in 2 weeks
                - may leave chronic vasculitis, especially in young/middle aged women
            - trench foot (immersion foot)
                - prolonged wet non-freezing condition <10°
            - frostbite
                - localised/extensive tissue necrosis
                - may require amputation
    - classification:
        - 1st degree
            - central whitish area with surrounding erythema
        - 2nd degree
            - clear/cloudy blisters within 24h
        - 3rd degree
            - haemorrhagic blisters/hard black eschars
        - 4th degree
            - tissue necrosis
    - management:
        - rewarm early to 40-42° (unless there is a chance that refreezing could occur)
        - NSAIDs during rewarming to prevent thrombosis
        - prostaglandin E1 for 24 hours
        - operate once necrotic margins defined

EnvC4 decompression sickness

- risk to compressed gas divers, aviators, astronauts and caisson (large watertight structures that allow work below waterline such as in bridge construction) workers
- pathophysiology
    - Boyle's law: at a constant temperature the absolute volume of a fixed mass of gas is inversely proportional to the absolute pressure
        - as pressure increases, volume decreases
    - Henry's law: at a constant temperature, the amount of gas that will dissolve in a liquid is proportional to the partial pressure of the gas over that liquid
        - more nitrogen dissolves at greater partial pressures
        - nitrogen is not metabolised so the content does not change in inhaled/exhaled gas
        - as the diver descends, more nitrogen dissolves into the blood (on-gassing)
        - it comes out of solution as the diver ascends (off-gassing)
        - nitrogen coming out of solution can form bubbles, particularly if divers ascend too fast or miss any stops, leading to decompression sickness
- decompression illness

    - comprises decompression sickness and arterial gas embolism
- decompression sickness
    - nitrogen coming out of solution can cause a mass effect and inflammatory response in the tissue
        - can be anywhere in the body but more common in articular cartilage and nervous tissue (particularly spinal cord)
    - two main effects, primary (direct damage to tissue from gas bubbles, likely due to artery occlusion, damage to vascular endothelium and venous outflow obstruction with a disturbance of vascular permeability and microvascular flow) and secondary (inflammatory response, activation of clotting cascade, platelets, complement and leucocytes)
    - can present from 0-72 hours or more after a dive (inflammatory responses present later
- arterial gas embolism
    - nitrogen bubbles escape into the arterial circulation
    - may occur from:
        - pulmonary barotrauma
            - occurs if a diver does not equalise pressure (holding breath on ascent) or has an underlying lung disease causing gas trapping
            - over inflation injury can occur and air from the pneumothorax can embolise to the pulmonary circulation and into the left heart
        - patent foramen ovale/arteriovenous malformation (right to left shunt)
        - overwhelming the pulmonary filter
            - bubbles ordinarily diffuse to the alveoli and are exhaled, but this filter can be overwhelmed (e.g. with exercise) so bubbles pass through the lungs without being filtered out and enter the arterial circulation via the left heart
    - escaped gas can lodge anywhere in the arterial system causing ischaemia
        - most serious is in the cerebral arterial circulation (cerebral arterial gas embolus, CAGE)
    - quick onset of symptoms, swift recompression required to reduce permanent damage
- presentation
    - 90% within 6 hours of a dive
    - common presentations:
        - limb or joint pain
            - often toothache like, not worse on movement or palpation
        - girdle pain
            - coming from back and spreading to abdomen
        - neurological symptoms
            - tingling, numbness, weakness, behavioural change, personality change, poor co-ordination, loss of bladder/bowel control, changes in hearing or vision, memory loss, unconsciousness
        - chest pain/breathing difficulties
            - could be pneumothorax, gas in coronary vessels, immersion pulmonary oedema
        - rash

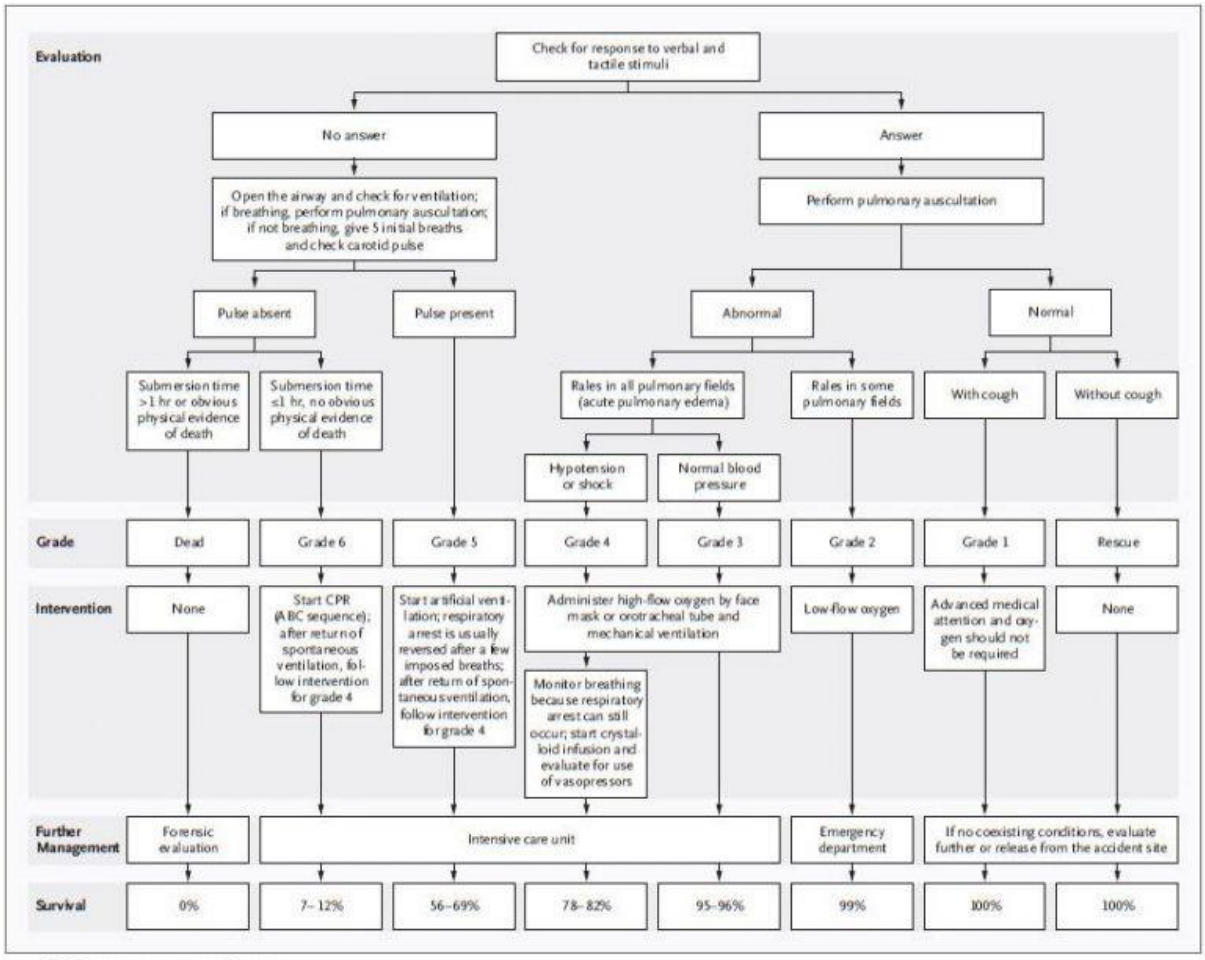
                - mottled, called cutis marmorata
                - can precede more severe neurological decompression illness
                - can disappear quickly and should be photographed
            - audio-vestibular
                - hearing loss, balance and co-ordination problems, vertigo, vomiting
                - difficult to differentiate from inner ear barotrauma
            - not quite right
                - malaise, headache, lethargy, loss of appetite, apathy, cognitive symptoms
- history
    - how deep?
    - how long?
    - any missed stops?
    - surface interval between dives
    - multiple dives (how many? over how many days? how many consecutive days?)
    - closed or open circuit and which gases
        - open most common, gas from a tank via hose and regulator; when diver exhales, gas from lungs goes into environment (bubbles); depth time limited by amount of gas in tank
        - closed circuit rebreather uses a scrubber so gas waste is limited and exhaled waste is partially removed; air is recycled and topped up by a small pony tank – usually oxygen, sometimes mixed gas; few or no bubbles whilst diving; more commonly used by specialist or military divers but may be used by recreational divers
            - less inert gas is inhaled through the dive so inert gas load will be less
        - semi-closed circuit rebreathers – rarely used by recreational divers
    - any problems during dive (e.g. rapid ascent)
    - any improvement with oxygen? – symptoms tend to improve, but not diagnostic
    - timing of symptoms
- investigations (primarily clinical diagnosis)
    - pulse oximetry
    - CXR (pulmonary barotrauma, surgical emphysema, tension pneumothorax, immersion pulmonary oedema)
        - consider CT chest if any uncertainty – barotrauma may need to be treated before recompression therapy starts
    - full neurological examination
    - joint examination
    - skin examination
- management
    - call National Diving Accident Helpline or Divers Alert Network
    - oxygen (even if sats are high)
        - 15l/min via non-rebreathe mask
    - CXR – chest drain if pneumothorax
    - fluids – low threshold for IV
    - consider catheter (urinary retention is common)
    - NEVER give Entonox to anyone who has recently dived
        - will worsen nitrogen load and barotrauma

    - NO painkillers unless very long transfer to chamber and only after discussion with dive doctor
        - NSAIDs can exacerbate micro-haemorrhages and have permanent sequelae
        - opiates can increase risk of oxygen toxicity (they will be exposed to high partial pressures during recompression and lowering toxicity threshold can precipitate seizures)
        - oxygen is often sufficient for analgesia anyway
    - recompression therapy
        - 3 main effects
            - bubble crushing (increased pressure decreases volume of bubbles)
            - flushing out nitrogen bubbles with oxygen
            - healing damaged tissue with hyperbaric oxygen

EnvC5 near-drowning

- submersion is when the airway goes below the level of the water, immersion is liquid splashed across a person's face; WHO definition of drowning is 'the process of experiencing respiratory impairment from submersion/immersion in liquid'
- WHO records accidental drowning rate of around 6.8 per 100,000 (second highest cause of death from injury after RTC)
- sequence of events:
    - waters enters the mouth when it cannot be kept clear
    - voluntarily spat out or swallowed
    - next response is breath holding
        - lasts around 1 minute until inspiratory drive too high to resist
    - water aspirated into airways, coughing occurs as reflex response
    - laryngospasm may occur (terminated by onset of brain hypoxia)
    - continued aspiration leads to hypoxaemia, loss of consciousness and apnoea
    - final mode of death involves cardiac dysrhythmia (tachycardia, bradycardia, PEA then asystole)
    - whole process usually occurs in seconds to a few minutes
- diving reflex
    - may provide an element of protection in cold water in young children
    - apnoea, bradycardia and peripheral vasoconstriction when the face contacts cold water
- cardiovascular caution
    - exiting water causes loss of the pressure effect of water (which was causing increased venous return and cardiac output) and can cause circulatory collapse – patients should be extricated horizontally if possible
- management:
    - patients who are asymptomatic and have no evidence of respiratory compromise (no CXR changes, no hypoxia on ABG) after 6 hours can be discharged home; all symptomatic patients should be admitted for observation
    - remove wet clothes, insulate with blankets, passive rewarming/active rewarming
    - NG tube to decompress stomach
    - neurovascular: head up, low normal CO_2, MAP 80mmHG, benzodiazepines for seizure, therapeutic hypothermia (34°)
    - respiratory: protective lung ventilation, bronchodilation, prone position, ECMO

    - metabolic: severe metabolic acidosis from lactate, PaO2 is much lower in a cold patient than on ABG as it is warmed to 37°, rhabdomyolysis
    - cardiovascular: VF secondary to hypothermia, hypovolaemia due to extravasation of systemic and pulmonary capillaries and cold diuresis
    - infection: consider antibiotics if submerged in grossly contaminated water
- predictors of poor outcome:
    - scene
        - immersion >10 minutes
        - delay to CPR (unwitnessed, no bystander CPR)
        - time to first breath
        - water temperature (drop in brain temp of 10° doubles time brain can survive)
        - presence of cardiac arrest
        - identifiable precipitants (e.g. arrest secondary to MI whilst in pool)
    - ED
        - asystole
        - CPR >25 minutes
        - dilated, non-reactive pupils and pH <7.0
        - dilated, non-reactive pupils and GCS <5
        - lactate
    - ICU
        - loss of grey-white matter differentiation on CT within 36 hours
        - absence of purposeful motor response (GCS <5) and absence of brainstem reflexes, pupillary response and spontaneous respiration at 24h


Evaluation
Check for response to verbal and tactile stimuli
No answer
Answer
Open the airway and check for ventilation; if breathing, perform pulmonary auscultation; if not breathing, give 5 initial breaths and check carotid pulse
Perform pulmonary auscultation
Pulse absent
Pulse present
Abnormal
Normal
Submersion time >1 hr or obvious physical evidence of death
Submersion time ≤1 hr, no obvious physical evidence of death
Rales in all pulmonary fields (acute pulmonary edema)
Rales in some pulmonary fields
With cough
Without cough
Hypotension or shock
Normal blood pressure
Grade
Dead
Grade 6
Grade 5
Grade 4
Grade 3
Grade 2
Grade 1
Rescue
Intervention
None
Start CPR (ABC sequence); after return of spontaneous ventilation, follow intervention for grade 4
Start artificial ventilation; respiratory arrest is usually reversed after a few imposed breaths; after return of spontaneous ventilation, follow intervention for grade 4
Administer high-flow oxygen by face mask or orotracheal tube and mechanical ventilation
Low-flow oxygen
Advanced medical attention and oxygen should not be required
None
Monitor breathing because respiratory arrest can still occur; start crystalloid infusion and evaluate for use of vasopressors
Further Management
Forensic evaluation
Intensive care unit
Emergency department
If no coexisting conditions, evaluate further or release from the accident site
Survival
0%
7–12%
56–69%
78–82%
95–96%
99%
100%
100%

Click image to enlarge

Referece: Szpilman D et al. N Engl J Med 2012;366:2102-2110. Treatment of Persons Who Have Drowned.

EnvC6 radiation exposure and safety

- pathophysiology:
    - radiation kills cells by disrupting neutral atoms, dislodging orbital electrons to form an ion pair consisting of a dislodged electron and the residual atom; the ion pairs are highly chemically active
    - rapidly dividing cells are more vulnerable
        - at lower doses, cells do not die but are at risk of DNA damage and malignant change
    - lethal dose to kill 50% of the population at 60 days is 4.5 sieverts
    - with medical management, 50% of the population would survive exposure to 5-4 sieverts
    - cumulative dose depends on:
        - measured dose at exposure
        - duration of exposure
        - rate of decay
        - distance from radioactive source (absorbed radiation decreased proportionally to the square of the distance)
        - shielding (e.g. lead)
- presentations:
    - local effects:

        - short term sequelae
            - erythema
            - desquamation (dry or wet)
            - blistering
            - oedema
            - pain
        - long term sequelae
            - vascular insufficiency
            - ulceration
            - necrosis
            - entry point for infection in potentially immunocompromised patient
    - systemic effects:
        - prodromal phase (lasts around 12 hours)
            - nausea
            - vomiting
            - weakness
            - fatigue
            - neurological signs
        - latent phase (lasts 5-7 days)
            - patient seems to recover
        - period of obvious illness
            - gingival bleeding
            - epistaxis
            - petechiae
            - systemic infections and gastrointestinal symptoms lasting up to 4 weeks
                - risk of infection highest at 25-35 days due to marrow suppression
        - recovery or death (final stage)
- effects according to exposure:
    - 1 Sv
        - mild or absent symptoms
        - episodic nausea and vomiting for 48h
        - mildly depressed WCC at 2-4 weeks
        - no foetal effects
        - counselling required if pregnant and >100mSv
    - 1-8 Sv
        - haematopetic syndrome
        - anorexia, nausea and vomiting, fatigue 1-4h after exposure
        - latent (2-28 days)
        - bone marrow suppression, leucopaenia, infection, thrombocytopaenia, bleeding, bruising; hair loss at 2-4 weeks
        - serial lymphocyte counts predicts severity
        - survival 50% without treatment, 60% with treatment
    - 6-20 Sv
        - gastrointestinal syndrome, early nausea and vomiting
        - fatigue and anorexia

        - latent hours – 1 week
        - severe GI symptoms: fever, abdominal pain, cramps, watery diarrhoea, haemorrhage, electrolyte imbalance, dehydration
        - also associated with bone marrow suppression
        - >10 Sv usually results in death within 2 weeks
    - >20 Sv
        - almost instant vomiting, explosive bloody diarrhoea, headache, collapse, decreased level of consciousness, agitation, burning sensation of skin
        - may have lucid interval in hours
        - death from coma, convulsions, hypotension, shock
        - death in a few days
- Acute Radiation Syndrome score
    - used by British institute of Radiology
    - grades involvement of four specific systems with scores from 1-4
    - neurovascular (N), haematological (H), cutaneous (C), gastrointestinal (G)
    - grading code generated with a severity index 'i', which can be used to generate a response category (RC) specific to a time point
        - used to guide further examinations and investigation and predict likelihood of recovery
    - lymphocyte count at 12 hours can also predict severity of exposure
- investigations:
    - radiation detection
        - Geiger counter
        - area survey meter
        - personal dose-rate meter
    - blood tests
        - FBC at 12 hours then every 4 hours
        - U&Es to assess level of dehydration and guide fluid therapy
- management:
    - general measures
        - preparation
            - consideration of command/control issues, nominating organisation responsibility, personnel training, notification criteria, equipment
            - CBRN training for senior medical and nursing staff in EDs
            - familiarity with hospital response plan and equipment
            - safety officer for radiotherapy department should be involved in a significant radiation incident
        - acute crisis management
            - how to triage and deal with injured patients
            - limit further exposure to population and personnel
            - organise decontamination and evacuation
        - management of long-term consequences
            - personal safety
                - at 0.1 mGy/h personnel can enter and give life-saving or time-critical treatment
                - at >0.1 mGy/h exposure is life threatening so personnel should not proceed

        - all staff dealing with contaminated patients should wear protective clothing and carry a personal radiation meter
    - initial assessment and triage
        - categorisation into life-threatening and non-life-threatening injuries
        - those with life-threatening injuries should be treated as contaminated and evacuated to a facility pre-alerted to their arrival
        - those with non-life-threatening injuries and the non-injured should be evacuated upwind and formally assessed for contamination
    - decontamination
        - not usually possible on scene if large numbers affected
        - up to 90% of contamination is removed by removing clothes
        - exposed individuals hosed down with warm water and detergent
        - should use rinse-wipe-rinse system and avoid skin abrasion
        - all contaminated materials and water should be disposed of appropriately
- specific measures
    - ED should be divided into areas for contaminated and non-contaminated patients
        - safe transfer between can be achieved by wrapping patient in a sheet, limiting cross-contamination
    - on arrival patients can be segregated into external contamination only, internal contamination only, combination injuries and trauma associated with detonation devices
    - external radiation injuries
        - rinse wounds with saline and leave open until debrided and decontaminated
        - surgical excision of long half-life materials may be required
        - wounds should then be closed or covered to prevent infection
        - analgesia, antibiotic prophylaxis and possible vasodilator therapy and referral for plastic surgery as appropriate
    - internal contamination
        - aim is to reduce overall dose by reducing absorption, dilution, blocking, displacing, increasing elimination, chelation and decorporation
        - strategy depends on particular substance (e.g. decorporation is the removal of internal contamination by exploiting chemical and biological properties of the radioisotope – Prussian blue for caesium, bicarbonate for uranium)
    - acute radiation sickness

        - supportive (IV fluids, antiemetics, analgesia, nutritional support, antibiotics, antifungals, antivirals, blood component substitution, reduction of brain oedema)
        - haematologists are best qualified to look after these patients as the condition is similar to aplastic anaemia
    - long term management
        - potassium iodide or iodate to prevent thyroid accumulation of radioiodine
        - GSF
        - GM-CSF
        - bone marrow transplant
        - stem cell therapy
        - definitive surgery
        - cancer surveillance
        - infertility and teratogenesis management
        - psychosocial care

EnvC7 industrial chemical incidents

- legislation:
    - industrial chemical use is regulated by the Control of Major Hazards Regulations 2015 (COMAH)
        - enshrined in the Seveso III directive from the EU
        - industries such as those using oil, gas, chemicals or explosives are reportable to COMAH
        - each company must have a major accident action plan and be involved in surveillance
    - the European Major Accident Hazards Bureau (MAHB) has a database of all reportable incidents
    - the International Programme on Chemical Safety (IPCS) is run by the WHO and is worldwide
    - HAZOPS is a risk assessment system balancing hazard and gain to a level called As Low as Reasonably Practicable (ALARP) – an acceptable standard in law
        - risk assessment takes into account centres of population, infrastructure and geographical factors such as watercourses and prevailing winds
    - the CCA (Civil Contingencies Act 2004) moved responsibility for decontamination from the fire service to the health service unless there are mass casualties involved
        - each acute health trust must have a regularly updated major incident plan
        - hospital priorities are:
            - containment of the incident and prevention of secondary contamination
            - triage with basic first aid
            - decontamination when it has not taken place on scene
            - recognition of toxidromes and seeking of advice
            - appropriate treatment – supportive or antidotal
            - transfer to definitive treatment
            - safe end to the hospital response

                - continuation of business after the event
            - it should go along the lines of guidance from Public Health England and the Department of Health
- information:
    - Public Health England has resources on specific chemicals on their website
    - Toxbase/NPIS
    - Hazchem (UK Hazard Identification System) manages chemicals and information
        - vehicles transporting chemicals have boards stating the possible hazard, the UN product number, emergency response codes for pre-hospital personnel and a 24 hour contact number
- history:
    - exposure to what and how much (volume and concentration)
    - route of exposure
    - protective measures taken
    - treatment given
- most poisons have no specific treatment and supportive care is given
    - in known paraquat poisoning, excess oxygen can increase long term pulmonary fibrosis (hypoxia is also bad)
- chemical weapons
    - often organophosphate based
    - have an antidote (atropine/pralidoxime) so important to recognise
    - use the mnemonic SLUDGE for systemic symptoms (alongside pinpoint pupils)
        - Salivation
        - Lacrimation
        - Urination
        - Diarrhoea
        - GI upset
        - Emesis
- management:
    - avoid contamination
        - three phases
            - primary
                - from the incident, can only be minimised before the event with safety measures, PPE, handling plans etc.
            - secondary
                - from contaminated people leaving the scene and taking the chemical with them
                - may occur from exhalation of certain patients (e.g. cyanide)
            - tertiary
                - environmental, including air and water borne spread
        - patients should be assessed from a distance outside the department
        - there must be a hot and cold area, and the patient can only cross between once decontaminated
        - staff must be aware of whether they are dirty or clean
    - 70-85% of chemical can be removed by taking off contaminated clothes
        - dispose of as hazardous waste

- for non-caustic chemicals dry decontamination should be used – blotting and rubbing exposed skin gently with dry absorbent material (e.g. blue roll)
    - bag all waste for disposal
- for caustic chemicals (e.g. with itching, redness of skin, burning eyes) wet decontamination is needed (except for chemicals such as sodium which react violently with water)
- do not neutralised an acid or base with the opposite – can cause exothermic reaction and burns

EnvC8 bites and envenomations typical for the UK

- adder bites
    - Vipera berus, the common European adder, is the UK's only venomous snake
    - around 100 patients a year present with adder bites
        - most occur in summer
        - half are from trying to pick the snake up
    - 70% of bites are 'dry' – no envenomation
        - if patients are well and asymptomatic at 4 hours after the bite, they can be discharged
    - local envenomation
        - bites are extremely painful
            - followed by paraesthesia and swelling, spreading proximally
            - looks like cellulitis, with swollen lymph nodes and lymphangitic lines
            - rapid spread
            - can rarely progress to compartment syndrome or necrosis
        - management:
            - immobilise with splint or sling
            - analgesia
            - observation
            - clean and evaluate wound, check for foreign bodies
            - check tetanus status
    - systemic envenomation
        - rapid onset of anaphylactoid symptoms within minutes
        - profound shock, airway-threatening angio-oedema
        - multi-organ failure can occur
        - coagulopathy can occur
        - management:
            - treat as anaphylaxis
            - airway and cardiovascular support
            - antivenom
    - indications for antivenom:
        - shock
        - signs of systemic envenomation (e.g. angioedema, acidosis)
        - local envenomation with rapidly progressive swelling (e.g. spreading past wrist or ankle within 4 hours)
        - local envenomation with significant swelling (e.g. >1/2 of limb) within 48 hours

    - antivenom is given as an IV infusion, 10ml diluted in 500ml saline (or 5ml/kg saline for children) over 30 minutes
        - can be repeated after 1-2 hours
        - for severe reactions two vials can be given straight away
        - reactions to antivenom are common, about 10% have an anaphylactoid reaction and need adrenaline, steroids and antihistamine (could consider given a small dose of adrenaline before the antivenom)
- Hymenoptera stings
    - includes bees, wasps and ants
    - wasp venom allergy is more common in the UK than bee venom allergy
    - risk of reaction is increased by 58% if patient was stung previously in past two months
    - large local reactions (area of induration 10cm which peaks between 24 and 48 hours) occur in a quarter of people
        - management is with antihistamines and consider oral prednisolone for large reaction
    - anyone with a systemic reaction (anaphylaxis) should be referred to an allergy specialist
        - venom immunotherapy is also recommended and lasts around 3 years
- Hemioptera (true bugs)
    - only medically significant species in the UK is bedbugs (Cimex lectularius)
    - no evidence of disease transmission
    - can cause sleeplessness and bites can be painful and swollen
    - can be found by searching bed at night or hiding places in the day
    - resemble lentils and can live for 6 months without feeding
    - require removal or steam cleaning of mattress and insecticide treatment of room
- Ixodoidea (ticks)
    - most common in UK are Ixodes ricinus (sheep tick) – vector of Lyme disease, and Ixodes hexagonus (hedgehog tick)
    - bites are usually painless but can cause local sensitisation and secondary infection
    - worldwide, can transmit Rocky Mountain spotted fever, Colorado tick fever, Lyme disease, Q fever, tick paralysis, tick typhus and others
    - remove with tick remover or by pressing the ends of small forceps down into the skin alongside the tick's head, gripping the head and applying steady traction perpendicular to the skin without twisting
        - if mouthparts are left in the wound use local anaesthetic and scrape away with scalpel blade
    - doxycycline for 10 days may be given in areas of significant Lyme disease

EnvC9 high altitude emergencies – cerebral and pulmonary oedema

- high altitude illness includes:
    - acute mountain sickness
        - rare below 2500m
        - similar to bad hangover
            - headache plus one or more of: nausea, vomiting, lassitude, dizziness, difficulty sleeping
        - risk factors include:

                - previous AMS, rapid ascent, higher altitudes, strenuous physical exertion
            - recent altitude exposure can be protective
            - those over 50 may be less prone
        - high altitude cerebral oedema (HACE)
            - potentially fatal
            - usually occurs over 3000m
            - estimated prevalence 0.5-1% between 4000 and 5000m
            - symptoms include:
                - severe headache, confusion, ataxia, drowsiness, stupor, coma
            - ataxia is most sensitive sign and should be an indication for descent
        - high altitude pulmonary oedema (HAPE)
            - leading cause of death related to high altitude
            - usually occurs within first 2-4 days of ascent
            - decreased exercise capacity, dry cough, cyanosis, dyspnoea at rest, pink frothy sputum
            - HAPE and HACE can occur together
        - high altitude retinal haemorrhage
            - more common over 5000m
            - usually asymptomatic and resolve over a few weeks
            - may be found in HAPE and HACE and in otherwise well people
            - central scotoma can occur if macula involved
- causes:
    - cause of AMS and HACE not fully understood
        - vasogenic mechanism thought to be responsible for cerebral oedema (hypoxia induced cerebral vasodilation and alteration of the permeability of cerebral capillaries)
        - cytotoxic oedema may be involved (failure of Na+/K+/ATPase due to oxygen radicals)
    - HAPE is caused by heterogenous hypoxia-induced pulmonary vasoconstriction
        - those prone to it have greater rises in pulmonary artery pressure at altitude
        - causes diversion of flow to less constricted areas with capillary leakage
        - diminished reabsorption of alveolar fluid likely to be important
- prevention:
    - slow ascent
        - no more than 500m/day above 3000m with a rest day every 3 to 4 days
    - AMS/HACE prevention
        - acetazolamide prophylaxis (125mg BD)
            - carbonic anhydrase inhibitor, causes a bicarbonate diuresis and metabolic acidosis
            - increases hypoxic ventilatory response, decreases CSF production, may effect peripheral chemoreceptors
            - usually well-tolerated
            - side effects include paraesthesia and metallic taste
        - dexamethasone prophylaxis (2mg QDS)
            - usually reserved for short exposures (<4 days) due to side effects
        - avoid over-exertion, alcohol, smoking

        - eat a high carb diet and have adequate hydration
        - drug prophylaxis should be continued until descent or until 3 days have been spent at peak altitude
    - HAPE prevention
        - nifedipine prophylaxis (30mg SR BD)
            - reduces pulmonary artery pressure
            - should only be used for individuals with a prior history of HAPE, started on day of ascent and continued until descent or 5 days spent at target elevation
            - phosphodiesterase inhibitors (sildenafil, tadalafil) can also be used
        - salmeterol increases alveolar fluid clearance through action on Na+ transport
- management:
    - mild AMS:
        - stop ascent for 24 hours
        - acetazolamide
        - simple analgesia
        - may continue after resolution, but there is a risk of recurrence
    - severe AMS or HACE:
        - descent
        - supplemental oxygen
        - portable hyperbaric chamber if available
        - acetazolamide and dexamethasone
    - HAPE
        - descent
        - supplemental oxygen
        - portable hyperbaric chamber
        - nifedipine
        - CPAP if available
    - patients with HAPE and HACE
        - can be given respective treatments simultaneously
        - altered mental state in a patient with HAPE may be due to hypoxia not HACE – start dexamethasone if neurological status does not improve with oxygen
        - nifedipine will decrease MAP, which will decreased cerebral perfusion pressure and may lead to cerebral ischaemia in a patient with HACE

EnvC10 acid attacks

- may be acid or alkali
- management:
    - pre-hospital:
        - irrigate with clean water as soon as possible
            - even dirty water is better than nothing
            - try to avoid it running onto unaffected areas
        - remove clothes, jewellery and anything else that might have the corrosive substance on it
        - analgesia
        - consider clingfilm to cover burns once decontaminated

- ED :
    - irrigate for as long as needed to return pH to normal
        - use litmus paper after initial irrigation and 5, 10, 30 minutes etc
    - assess for airway involvement
    - prioritise eyes
        - use topical tetracaine to facilitate prolonged irrigation
        - involve ophthalmology
            - will need topical antibiotics, steroids, artificial tears
    - consider trauma team activation
        - thorough primary, secondary and tertiary survey
        - include stab check (burns may be distracting)
    - if extensive burns use Parkland formula for fluid replacement
    - specialist involvement (e.g. plastics, burns centre)
    - psychological support

GASTROENTEROLOGY AND HEPATOLOGY

GP1 abdominal and loin pain

- aetiology
    - gastro-intestinal
        - oesophagitis
        - gastritis
        - peptic ulcer disease
        - gallstone disease
        - pancreatitis
        - bowel obstruction
        - appendicitis
        - diverticular disease
        - inflammatory bowel disease
        - ischaemic bowel
        - gastroenteritis
        - acute liver failure
        - incarcerated hernia
        - constipation
    - gynaecological
        - ectopic pregnancy
        - pelvic inflammatory disease
        - ruptured ovarian cyst
    - urological
        - renal colic
        - pyelonephritis
        - urinary tract infection
        - testicular torsion
        - epididymo-orchitis
    - vascular
        - abdominal aortic aneurysm

    - medical
        - myocardial infarction
            - ECG if any suspicion
        - pneumonia
            - consider CXR, especially if hypoxic
        - diabetic ketoacidosis
            - always check blood glucose +/- VBG/ABG
        - hypercalcaemia
            - other features may include vomiting, weight loss, change in bowel habit, anorexia
        - mesenteric adenitis
    - 40% will have a final diagnosis of non-specific abdominal pain
- pathophysiology
    - visceral pain
        - the autonomic nerve supply innervates organs and when they are stretched vague, poorly localised pain is generated
        - if the organs are affected by peristalsis, the pain can appear intermittent or colicky
        - organs are innervated bilaterally so pain is often felt in the midline even if the organ is not in the midline
        - visceral pain is normally localised by the patient to the embryonic site of the organ, which may be different to the actual site
            - foregut structures (e.g. stomach, liver) cause upper abdominal pain
            - midgut structures (e.g. small bowel, appendix) cause central abdominal pain
            - hindgut structures (e.g. colon, GU system) cause lower abdominal pain
    - parietal (somatic) pain
        - due to irritation of the parietal peritoneum and is well localised to the site of the organ
    - referred pain
        - may be felt at a distant side due to misinterpretation of stimuli by the brain based on embryonic development of the nervous system (e.g. shoulder tip pain from diaphragmatic irritation)
        - caution for referred pain when examination of the area is completely normal
- history
    - onset of pain (sudden or gradual)
        - sudden
            - perforation of viscus
            - peptic ulcer
            - ectopic pregnancy
            - rupture of aneurysm
            - impaction of a stone
    - duration or recurrence of pain
        - recurrent pain
            - peptic ulcer disease
            - renal colic

        - gallstone colic
        - diverticulitis
        - Mittelschmerz
        - endometriosis
    - character or nature of pain
        - sharp, constant pain worsened by movement may suggest peritonitis
        - pain more marked than physical findings suggests ischaemic bowel or pancreatitis
        - pain due to inflammation of an organ tends to come on gradually and results in guarding on examination
        - pain due to obstruction tends to be intermittent and come in waves
    - location of pain
        - pain radiating to the back may represent aortic aneurysm rupture, peptic ulcer disease or pancreatic pathology
- associated symptoms
    - nausea and vomiting
        - vomiting is non-specific and present with many causes of abdominal pain
        - pain followed by vomiting suggests a surgical cause
    - altered bowel habit
        - diarrhoea
        - constipation
        - rectal bleeding
        - melaena
    - urinary symptoms
        - dysuria
        - frequency
        - haematuria
    - gynaecological history
        - last menstrual period
        - vaginal bleeding
        - vaginal discharge
        - dyspareunia
    - genito-urinary medicine
        - sexual history may be important and should be asked about if relevant
- management
    - resuscitation as required
    - morphine IV titrated to effect
    - no evidence for antispasmodics such as buscopan in the management of acute pain
    - IV anti-emetics – cyclizine and ondansetron preferred as metoclopramide theoretically increases gastric emptying (poor evidence base)
    - anti-pyretic if needed
    - NG tube if bowel obstruction present
    - urinary catheter if patient unwell or peritonitis suspected
    - broad spectrum IV antibiotics if signs of sepsis or peritonitis
    - nil by mouth and IV fluids
    - referral to surgical team

    - patients requiring morphine will generally require a period of observation as pain may recur once morphine wears off
    - stable patients may be discharged with safety netting +/- outpatient follow up
- surgical management options
    - wait and see
        - serial clinical examinations over time, preferably by senior surgeon
    - wait and image
        - observe and re-examine
        - US/CT/endoscopy
    - look and see
        - diagnostic laparoscopy
        - particularly useful in women of childbearing age at risk of tubo-ovarian pathology
    - open up
        - specific procedure depending on diagnosis
        - diagnostic laparotomy if cause unknown but surgical cause remains likely
        - conditions requiring operative intervention include:
            - presence of peritonism
            - frank peritonitis
            - evidence of perforation
            - failure to settle with non-operative measures
        - an exploratory laparotomy will give a cause in around 80%
- functional and chronic abdominal pain
    - any patient representing within a few days is at high risk of significant pathology
    - for patients who reattend frequently, it is important to take every presentation at face value
    - functional abdominal pain should be considered in patients with multiple presentations and previously normal investigations
    - social and medical input may be required +/- referral to chronic pain or mental health teams

GP2 abdominal swelling or mass

- background
    - masses are usually detected on physical examination rather than presented by the patient
- examination
    - supraclavicular and inguinal nodes
    - inspection
        - scars
        - distension
        - prominent veins
        - local swelling
        - pulsation
        - visible peristalsis
        - skin lesions
        - asymmetrical movement at eye level
        - exclude lesions of the abdomen

                - patient raises head
                - patient does straight-leg raising (Carnett's method)
                - 'blowing test' (Valsalva)
                - patient strains as if toileting (Kamath's test)
        - palpation
            - light, then deep
            - guarding, rigidity, rebound tenderness
            - mass:
                - site
                - tenderness
                - size and shape
                - surface (irregular or smooth)
                - edge (regular or irregular)
                - consistency (soft or hard)
                - mobility
                - pulsatile
                - ballotable
- causes of abdominal mass by location
    - right upper quadrant
        - cholecystitis
            - very tender mass
        - cholangiocarcinoma
            - moderately tender, irregularly shaped mass
        - hepatomegaly
        - liver cancer
            - firm, lumpy mass
    - epigastric
        - hepatomegaly
            - firm, irregular mass
        - pancreatic abscess or pseudocyst
        - gastric carcinoma
    - left upper quadrant
        - splenomegaly
        - gastric carcinoma
        - pancreatic abscess or pseudocyst
        - disorders of kidney or colon
        - neurofibroma (rare)
    - right flank
        - hydronephrosis
            - smooth spongy mass
        - renal cell carcinoma
            - smooth, firm, non-tender
    - periumbilical
        - abdominal aortic aneurysm
            - pulsating mass
        - gastrointestinal tract tumour
    - left flank

        - hydronephrosis
        - renal cell carcinoma
    - right iliac fossa
        - actinomycosis
        - amoebic abscess
        - appendix mass or abscess
        - caecal/colon cancer or distension
        - Crohn's disease
            - multiple tender, sausage-shaped masses
        - hernia
        - ileocaecal mass caused by tuberculosis
        - intussusception
        - kidney abnormality
        - ovarian tumour
        - tumour in intra-abdominal testicle
    - suprapubic
        - distended bladder
        - neuroblastoma
            - children and infants
    - left iliac fossa
        - diverticular abscess
        - hernia
        - kidney abnormality
        - ovarian tumour
        - colorectal cancer
        - tumour in intra-abdominal testicle
    - pelvis (not possible to palpate below mass)
        - ovarian cyst
            - smooth, round, rubbery mass
        - ovarian tumour
        - pregnancy
        - uterine fibroids (round lumpy mass) or malignancy
- investigations
    - depend on site and suspected diagnosis
    - may include:
        - US or CT
        - contrast medium for hollow organs (e.g. barium enema, gastrointestinal series, IV pyelogram)
        - FBC, ESR/CRP, U&E, LFT
        - CXR/AXR
        - US or CT-guided fine needle biopsy
        - Mantoux test
        - paracentesis with fluid examination if ascites present
        - laparoscopy or laparotomy may ultimately be required

GP3 ascites

- background

    - excessive accumulation of fluid in the abdominal cavity
    - to be detectable by clinical examination there needs to be at least 1500ml present (slightly less in a small, thin person, more in an obese person)
    - ultrasound can detect much smaller volumes (<500ml)
- grading
    - grade 1
        - mild ascites, only detectable by ultrasound
    - grade 2
        - moderate ascites causing moderate symmetrical distension of the abdomen
    - grade 3
        - large ascites causing marked abdominal distension
    - refractory ascites can be divided into two groups:
        - diuretic-resistant ascites is refractory to dietary sodium restriction and intensive diuretic treatment for at least one week
        - diuretic-intractable ascites is refractory to therapy due to the development of diuretic-induced complications that preclude the use of an effective dose of diuretic
- causes
    - cirrhosis
        - ascites is the most common manifestation and associated with reduced survival rate
        - around 50% of patients with cirrhosis develop ascites over a 10 year period
        - fluid retention (including ascites, peripheral oedema and pleural effusions) is the most frequent complication of end-stage ascites
            - significantly impairs quality of life and has poor prognosis
        - in patients with stable cirrhosis who suddenly develop ascites, hepatocellular carcinoma must be excluded
    - malignancy
        - accounts for 15% of cases
        - usual causes are:
            - malignancies of the gastrointestinal tract (carcinoma of stomach, colon, pancreas, primary hepatocellular carcinoma, metastatic liver cancer)
            - carcinoma of ovary (Meig's syndrome, which produces ascites out of proportion to the tumour and pleural effusions, often unilateral)
            - Hodgkin's and non-Hodgkin's lymphoma
            - metastatic lymphoma within the abdominal cavity
    - heart failure
    - nephrotic syndrome
    - protein-losing enteropathy
    - tuberculosis
    - pancreatitis
    - other rare causes, including hypothyroidism
    - iatrogenic (e.g. ovarian hyperstimulation syndrome)
- presentation
    - abdominal distension
    - weight gain as a result of water retention

    - discomfort
        - tense ascites is very uncomfortable
        - prior to this there is distension with mild discomfort
        - malignancy-related ascites is frequently painful
    - nausea and appetite suppression
        - tense ascites presses on the stomach and bowel
    - increasing dyspnoea
        - limited venous return from the lower limbs (pressure on the inferior vena cava)
        - impaired expansion of the lungs (pressure on the diaphragm)
    - other symptoms related to the cause of the ascites
    - risk factors for liver disease:
        - alcohol consumption
        - history of jaundice
        - history of chronic hepatitis B or C or risk factors for them
        - obesity, hypercholesterolaemia, type 2 diabetes (can cause non-alcoholic steatohepatitis, which can progress to cirrhosis)
- examination
    - shape of abdomen – flanks full on lying, fluid accumulates in lower abdomen on standing
    - high intra-abdominal pressure may push out umbilical or even inguinal hernia
    - signs of liver disease and cirrhosis
        - jaundice
        - muscle wasting
        - gynaecomastia
        - spider naevi
        - palmar erythema
    - rarely, a firm nodule in the umbilicus (Sister Mary Joseph's nodule) which suggests peritoneal carcinomatosis originating from gastric, pancreatic or hepatic primaries
        - check for left-sided supraclavicular node
    - shifting dullness
        - probability of ascites without shifting dullness is <10%
    - fluid thrill of large ascites
- monitoring
    - serial measurements of girth with tape measure in same position each time
    - serial measurement of weight
- investigations
    - aims
        - confirm presence of ascites
        - find cause of ascites
        - assess complications due to ascites
    - bloods
        - FBC
        - U&Es
        - LFTs
        - clotting
        - TFTs
        - other tests to assess cause, e.g. hepatitis serology

    - imaging studies
        - abdominal ultrasound
            - confirms ascites
            - may show causative pathology, e.g. ovarian carcinoma, metastatic liver disease
        - CXR
            - pulmonary effusion
            - pulmonary metastases
            - heart failure
        - MRI if ultrasound has not revealed a cause
    - tapping of ascites
- management
    - treatment of any underlying cause
    - salt intake limited to <90mmol/day (5.2g/day)
        - useful in cirrhosis, unlikely to be helpful in other aetiologies
    - drugs
        - diuretics
            - spironolactone is the best initial choice
                - increase sodium excretion and potassium reabsorption in the distal tubules
                - 100mg/day, gradually increased to 400mg as needed
                - serum potassium levels need to be monitored, risk of hyperkalaemia
            - loop diuretics
                - may be used as an adjunct, generally when maximum doses of spironolactone have been reached
                - start with 40mg/day furosemide, going up to 160mg/day
                - high doses cause severe electrolyte imbalance, particularly hyponatraemia
    - therapeutic paracentesis
        - for large or refractory ascites
        - sterile procedure
        - paracentesis of <5 litres of uncomplicated ascites should be followed by plasma expansion with a synthetic plasma expander
        - larger-volume paracenteses should be followed by volume expansion using human albumin solution
    - catumaxomab
        - trifunctional bispecific monoclonal antibody
        - intraperitoneal catumaxomab is useful for patients with malignant ascites secondary to epithelial cancers (especially gastric cancer) with an acceptable safety profile
    - surgical
        - transjugular intrahepatic portosystemic shunt (TIPS)
            - can be used for patients with refractory ascites needing frequent paracentesis (<3/month)
            - done with sedation and local anaesthetic

                - blockage occurs in around a quarter of cases; covered stents improve long term patency
- complications
    - hyponatraemia on diuretics
    - spontaneous bacterial peritonitis
        - occurs in 10-30% of people with ascites and has a mortality rate of 20%
        - frequently asymptomatic, may have fever, mild abdominal pain, vomiting, confusion
        - should be suspected in patients presenting with hepatic encephalopathy, renal impairment or peripheral leucocytosis without any obvious precipitating factor
        - organisms are usually E. coli, streptococci and enterococci
        - empirical antibiotics should be started if ascitic fluid contains >250 cells/mm^3
        - patients with SBP should be referred for consideration of liver transplantation
    - hepatorenal syndrome
- prognosis
    - patients with cirrhosis who develop ascites have a one-year mortality rate of 15% and a five year survival rate of 44%
    - for most patients with cirrhosis, therapeutic paracentesis and TIPS without transplantation may improve quality of life but are not thought to significantly improve long term survival
    - malignant ascites tends to suggest widespread disease and a poor prognosis
- paracentesis
    - indications
        - diagnostic (ascitic tap or paracentesis)
            - new-onset ascites
                - determine aetiology
                - differentiate transudate vs exudate
                - detect cancerous cells
            - suspected spontaneous or secondary bacterial peritonitis
        - therapeutic (usually via paracentesis)
            - relieve respiratory distress or abdominal pain resulting from ascites
    - contra-indications
        - uncooperative patient
        - skin infection at proposed puncture site
        - pregnancy
        - severe bowel distension
        - coagulopathy (may be considered relative contraindication)
    - investigations
        - prior to tap
            - FBC and clotting screen
                - consider pooled platelets for thrombocytopaenia or fresh frozen plasma if evidence of coagulopathy
            - U&Es, LFTs
            - abdominal ultrasound

        - not always required prior to tap
        - used to review liver, spleen, pancreas and lymph nodes
        - may also show causative pathology
    - after the tap
        - microscopy, WCC, red cell count, Gram stain
            - neutrophil count >250 cells/mm^3 is diagnostic of SBP
            - red cell count is usually <1000 cells/mm^3
                - higher levels raise the suspicion of an underlying malignancy
            - Gram stain is quick but rarely helpful
                - samples should also be sent for culture and sensitivity
                    - they should be inoculated into blood culture containers as soon as the sample is taken
        - albumin or protein levels
            - serum ascites-albumin gradient (SA-AG)
                - SA-AG ≥11g/L – likely causes include cirrhosis and cardiac failure
                - SA-AG <11g/L – likely causes include nephrotic syndrome, malignancy, pancreatitis and tuberculosis
        - amylase
            - will be higher in pancreatitis-associated ascites
        - cytology
            - yield is greater with higher volume samples (>100ml)
- risks
    - relatively safe procedure
    - complications more likely when comorbidities present
    - risk of serious complications is around 1 in 100 for minor complications and less than 1 in 1000 for major complications
    - risks include:
        - significant bleeding
        - infection
        - renal failure
        - hyponatraemia
        - hepatic encephalopathy
        - complicated bowel perforation
        - paracentesis leak
- technique
    - equipment
        - needles (25G for infiltration, 22G for fluid collection), syringes and local anaesthetic (may not be necessary for a tap)
        - antiseptic skin preparation (value unproven) and drapes
        - very wide bore IV cannula, IV giving set and a urine bag of the type attached to a catheter
        - adhesive tape
        - surgical gloves
    - explain the procedure and risks and obtain consent

        - position the patient, usually in the supine position with the head of the bed elevated to allow fluid to accumulate in the patient's lower abdomen
        - position of the tap:
            - locate area of flank dullness lateral to the rectus abdominis muscle and go approximately 5cm superior and medial to the anterior superior iliac spines
            - avoid the inferior epigastric vessels which run up the side of the rectus abdominis to anastomose with the superior epigastric vessels coming down
            - avoid the pelvic area, solid tumour masses, prominent superficial veins and scars (may have collateral vessels close by or adherent bowel beneath)
            - using local anaesthetic if needed, the needle is inserted and fluid aspirated
            - if this does not work, US guidance may help, particularly for small volume ascites
            - 10-20ml can be aspirated for diagnostic purposes
            - if a therapeutic tap is required, an IV cannula is placed using the Z-track technique which involves puncturing the skin perpendicularly and advancing the needle obliquely in the subcutaneous tissue
                - reduces leakage following the procedure as the puncture site on the skin and the peritoneum are not adjacent
            - once the cannula is in place, the needle is withdrawn and a giving set and collecting bag connected
                - drain for 6-8 hours then remove the cannula and cover with a simple adhesive bandage
            - swift drainage is safest but may need to be slowed or stopped if the patient develops hypotension
            - large volumes taken off over 2-4 hours can reduce intra-abdominal and inferior vena cava pressure and cardiac output may increase in response; this may lead to a reduction in blood pressure and colloid replacement is usually given
    - post-paracentesis circulatory dysfunction
        - withdrawal of 5 litres or more of ascites can precipitate post-paracentesis circulatory dysfunction:
            - hepato-renal syndrome and hyponatraemia
            - acute kidney injury
            - increased plasma renin activity
        - guidelines suggest that if >5 litres drained, albumin (20% or 25% solution) should be infused at a dose of 8g albumin per litre of ascites drained
        - there is no evidence that albumin or artificial plasma expanders prevent complications or improve outcome

GP4 constipation

- background
    - acute constipation is intestinal obstruction until proven otherwise
- red flags

    - weight loss
    - rectal bleeding/melaena
    - nausea/vomiting
    - fever
    - rectal pain
    - change in stool calibre
- differential
    - behaviour-related
        - lack of exercise
        - diet-related
    - faecal impaction
    - stercoral colitis (inflammatory colitis caused by impacted faecal matter distending the colon)
    - diverticulitis
    - bowel obstruction (tumour, stricture, hernia, adhesion, volvulus)
        - small bowel obstruction
        - large bowel obstruction
    - painful anorectal disorders
    - medical causes
        - hypothyroidism
        - hypomagnesaemia
        - hypercalcaemia
        - hypokalaemia
    - medication-related
        - opioids
        - antipsychotics
        - anticholinergics
        - antacids
        - antihistamines
- investigations
    - PR examination
    - bloods (electrolytes, TFTs)
    - CT abdo/pelvis if obstruction suspected
- management
    - adequate fluid (1.5 litre/day)
    - fibre (10g/day)
    - exercise
- medication options
    - emollient
        - docusate – facilitates mixture of stool fat and water
        - mineral oil – long term use causes malabsorption
    - stimulants
        - bisacodyl
        - senna
    - saline laxative
        - milk of magnesia
        - magnesium citrate
    - hyperosmolar agents

        - lactulose
        - PEG
        - glycerin suppositories
    - enemas
        - soap suds, saline, tap water (rectal distension causes evacuation)
        - phosphate enema
            - no more than 2 doses in 24 hours without bloods
            - may cause hyperphosphataemia, hypocalcaemia, hypomagnesaemia
            - high risk patients: renal impairment, abnormal gut motility, IBD, elderly, cardiac comorbidities

GP5 diarrhoea

- types
    - secretory
    - motor
    - exudative
    - osmotic
- causes
    - infective
        - pseudomembranous colitis (C diff)
        - Staphylococcal enterocolitis and typhlitis
        - Campylobacter spp
        - Clostridium perfringens
        - Salmonella spp
        - Shigella spp
        - Escherichia coli
        - Yersinia spp
        - Entamoeba histolytica
        - HIV
    - non-infective
        - enteral feed associated
        - ischaemic colitis
        - chemical colitis
        - gut dysmotility
        - hypoalbuminaemia
        - acute exacerbation of Crohn's or ulcerative colitis
    - other risk factors
        - fever or hypothermia
        - presence of infection
        - malnutrition
        - hypoalbuminaemia
        - sepsis
        - multiorgan dysfunction
        - previous TPN
- consequences
    - malnutrition
    - haemodynamic instability

    - metabolic acidosis (loss of electrolytes and bicarbonate)
    - hypokalaemia
    - hypomagnesaemia
    - low zinc
    - impaired wound healing
    - arrhythmias
- management
    - IV rehydration and correction of electrolytes
    - antibiotics for certain infectious causes as per guidelines
    - consideration of loperamide or codeine
    - consideration of probiotics or prebiotics
    - treatment of any underlying cause

GP6 haematemesis and melaena

- causes
    - peptic ulcer disease (75% gastric rather than duodenal)
    - varices (90% oesophageal rather than gastric)
    - oesophagitis
    - gastritis
    - duodenitis
    - Mallory-Weiss tears
    - portal hypertensive gastropathy
- investigations
    - bloods
        - FBC
        - coagulation
        - blood gas if unstable
    - other investigations as appropriate
    - consider H pylori testing
    - upper GI endoscopy
        - urgent if:
            - syncope
            - haematemesis (suggests stomach is filling with blood)
            - hypotension
            - transfusion requirements in excess of 4 units over 12 hours
            - age over 60
            - multiple comorbidities
- management
    - resuscitation
    - transfusion if needed with avoidance of under- and over-transfusion
    - correction of underlying bleeding diathesis
    - temporising balloon tamponade if indicated for variceal bleed
    - scoring systems:
        - Glasgow-Blatchford
            - consider discharge if score is 0
        - Rockall after endoscopy
    - hold PPI until after endoscopy

- variceal bleed
    - terlipressin
    - prophylactic antibiotics
    - TIPS if endoscopy unsuccessful

GP7 jaundice

- normal physiology
    - normal haem metabolism has three stages: pre-hepatic, hepatic and post-hepatic
    - pre-hepatic
        - as red blood cells reach the end of their lifespan (120 days) they pass through the reticulo-endothelial system in the spleen
        - RBCs are broken down by spleen macrophages into haemoglobin, which is further broken down into haem and globin
        - the globin portion is a protein which breaks down into amino acids
        - the haem portion is initially oxidised to form biliverdin then reduced to form unconjugated bilirubin
        - unconjugated bilirubin travels to the liver bound to serum albumin
        - the majority of bilirubin is produced from the catabolism of haem, with 20% from other haem sources such as ineffective erythropoiesis, the breakdown of myoglobin in muscle and the breakdown of cytochromes and catalase
    - hepatic
        - in the liver, an enzyme (UDP glucuronyl transferase) conjugates the bilirubin with glucuronic acid to form conjugated bilirubin
    - post-hepatic
        - conjugated bilirubin is excreted in the bile into the small intestine
        - enzymes within the intestine convert conjugated bilirubin to stercobilinogen and urobilinogen
        - stercobilinogen converts to stercobilin and is excreted through faeces – it is responsible for the colouration of faeces
        - urobilinogen is filtered by the blood and transported to the kidneys where it is oxidised to form urobilin, which is excreted through urine and is responsible for the colour of urine
- pathophysiology
    - pre-hepatic
        - any process that causes an increased rate of RBC breakdown (haemolysis) and saturation of enzymes can cause jaundice
            - malaria
            - sickle cell anaemia
            - spherocytosis
            - glucose-6PD deficiency
            - haemolytic uraemic syndrome
            - transfusion reaction
    - hepatic
        - any process which affects liver function can cause jaundice
            - drugs/toxins
                - alcohol
                - paracetamol

                - anabolic steroids
                - isoniazid
                - amanita toxin
                - chlorpromazine
                - flucloxacillin
                - halothane
            - infections
                - viral hepatitis
                - infectious mononucleosis
                - leptospirosis
            - metabolic
                - Wilson's disease
                - Reye's disease
                - haemochromatosis
            - granulomatous
                - Wegener's granulomatosis
                - lymphoma
                - sarcoidosis
                - mycobacterial
            - genetic
                - Gilbert's syndrome
                - Crigler-Najjar syndrome
                - Dubin-Johnson syndrome
            - other
                - fatty liver of pregnancy
                - primary biliary cirrhosis
                - amyloidosis
                - metastatic carcinoma
            - neonatal jaundice
    - post-hepatic
        - any process which causes post-hepatic obstruction can cause jaundice
            - drugs
                - amitriptyline
                - prochlorperazine
                - verapamil
                - co-amoxiclav
            - gallstones
            - pancreatic carcinoma
            - primary sclerosing cholangitis
            - biliary atresia
            - bile duct strictures
            - cholangiocarcinoma
            - pancreatitis
            - pancreatic pseudocyst
- history
    - alcohol intake
    - transfusion of blood products

    - sexual contact with a person known to have hepatitis or promiscuous sexual activity
    - intravenous drug misuse
    - recent tattoos or body piercings
    - recent foreign travel
    - needle stick injury
    - colour of urine and stool
    - weight loss
    - family history of jaundice
- examination
    - metastatic disease
        - hard nodular liver on a background of known malignancy
    - alcoholic liver disease
        - palmar erythema
        - spider naevi
        - proximal muscle wasting/weakness
        - hepatic flap
        - foetor hepaticus
        - cerebellar signs
        - encephalopathy
    - acute cholangitis
        - Charcot's triad
            - jaundice
            - fever
            - right upper quadrant tenderness
    - malignancy
        - painless jaundice and cachexia and an epigastric mass
- investigation
    - urine
        - pre-hepatic hyperbilirubinaemia
            - unconjugated bilirubin is bound to albumin and is not water soluble so cannot appear in the urine
            - if urine is negative for bilirubin, total bilirubin is raised and direct bilirubin is not raised, investigate for unconjugated hyperbilirubinaemia, haemolysis, drug toxicity, genetic disorder
        - post-hepatic hyperbilirubinaemia
            - conjugated bilirubin is water soluble and appears in the urine
            - urobilinogen is absent due to the inability of conjugated bilirubin to be excreted into the small intestine
            - if urine is positive and direct bilirubin is raised, investigate for conjugated hyperbilirubinaemia
    - LFTs
        - alkaline phosphatase (ALP)
            - normal range 25-115 U/L
            - considerably raised in extrahepatic and intrahepatic biliary disease
        - transaminases
            - AST 10-40 U/L
            - ALT 5-40 U/L

        - usually highly raised in hepatocellular disease
    - gamma glutaryl transferase (GGT)
        - male <50 U/L
        - female <32 U/L
        - sensitive but not specific for alcohol intake
        - raised GGT and ALP suggest cholestasis
- other bloods
    - FBC
        - raised reticulocyte count, schistocytes on blood film and positive Coombs' test indicates haemolysis
    - amylase
        - may indicate pancreatitis
    - hepatitis serology
    - autoimmune markers
        - anti-nuclear antibodies
        - anti smooth muscle antibodies
        - the majority of patients with PBC have anti-mitochondrial antibody; up to 85% have anti-nuclear cytoplasmic antibody
    - alpha-1 antitrypsin
        - deficiency may precede liver cirrhosis
    - copper/ceruloplasmin
        - Wilson's disease
    - ferritin
        - levels >1000ng/ml and transferrin saturations >50% indicates haemochromatosis
- imaging
    - abdominal ultrasound
        - useful to distinguish between hepatocellular and extra-hepatic causes
        - limited at detecting intraparenchymal disease of the liver or pancreas and is operator-dependent
    - CT
        - good at determining intraparenchymal liver and pancreas disease
    - magnetic resonance cholangio-pancreatography (MRCP)
        - non-invasive, radiation-free diagnostic test
        - useful for evaluating the biliary tree, pancreas and liver
        - findings determine whether an ERCP is required
    - endoscopic retrograde cholangio-pancreatography (ERCP)
        - may be required to detect disease in the biliary and pancreatic ducts
        - may be used therapeutically to remove gallstones from the biliary tract or to place stents across narrow ducts
        - most common and serious complication is pancreatitis
    - liver biopsy
        - used when serum and radiological investigations fail to provide a definitive diagnosis
        - particularly useful for diagnosing autoimmune hepatitis or biliary tract disorders

- management
    - ED involvement for jaundice is often limited to facilitating further investigations and an admission/discharge decision
    - jaundice can represent a medical emergency, including:
        - ascending cholangitis
        - fulminant hepatic failure
        - massive haemolysis
        - neonatal jaundice
    - patients with raised ALP and GGT are likely to have biliary tract obstruction
        - should have US requested in the ED and appropriate surgical/GI referral depending on the findings
    - patients with raised AST/ALT have hepatocellular injury and should be admitted if there is evidence of:
        - coagulopathy
        - sepsis
        - altered mental status
        - intractable pain/vomiting
            - otherwise outpatient investigation may be appropriate
    - patients with haemolysis should be admitted
- acute liver failure
    - patients with hepatocellular injury, coagulopathy and altered mental status may have acute liver failure/fulminant hepatic failure
    - these patients require fluid resuscitation, haemodynamic monitoring and critical care admission
    - most common causes are paracetamol poisoning (UK) and acute hepatitis B (worldwide)
    - prior to liver transplantation, mortality was 80%
    - indications for consideration of liver transplant after paracetamol overdose are:
        - acidaemia (pH <7.3)
        - renal insufficiency
        - hepatic encephalopathy
        - elevated PT

GP8 anal pain and rectal bleeding

- pain
    - bleeding
        - external haemorrhoid
        - prolapsed internal haemorrhoid
        - anal fissure
            - off midline – consider cancer, HIV, TB, Crohn's
    - no bleeding
        - swelling
            - anorectal abscess
                - perirectal
                - ischiorectal
                - intersphincteric
                - supralevator

                - pilonidal cyst
                - anal fistula
                - hidradenitis suppurativa
            - no swelling
                - proctalgia fugax
- no pain
    - bleeding
        - cancer
        - internal haemorrhoid
    - swelling
        - itch
            - condyloma acuminata
        - no itch
            - rectal prolapse
    - itching
        - discharge
            - proctitis
        - no discharge
            - pruritus ani
- lower GI bleed
    - type of blood
        - haematochezia
            - bright red or maroon coloured, comes from the rectum
            - usually represents lower GI bleed
            - may represent UGIB with brisk bleeding (usually with haematemesis and haemodynamic compromise)
        - melaena
            - usually represent UGIB
            - may represent slow bleeding from lower GI source
    - differential for lower GI bleeding
        - upper GI bleeding
        - diverticular disease
            - diverticulosis
            - diverticulitis
        - vascular ectasia/angiodysplasia
        - inflammatory bowel disease
        - infectious colitis
        - mesenteric ischaemia/ischaemic colitis
        - Meckel's diverticulum
        - colorectal cancer/polyps
        - haemorrhoids
            - internal
            - external
        - aortoenteric fistula
            - nearly 100% mortality if untreated
            - consider in patients with GI bleeding and known AAA or aortic grafts
        - rectal foreign body

        - rectal ulcer (HIV, syphilis, STI)
        - anal fissure
        - marathon runners
            - 16% have haematochezia within 24-48 hours of a race (not actionable unless abdominal pain present)

GP9 nausea and vomiting

- differential
    - GI
        - peptic ulcer disease
        - obstruction
            - adhesion
            - small bowel obstruction
                - SMA syndrome
            - gastric outlet obstruction
            - gastric volvulus
            - bezoar
        - pancreatitis
        - gastroparesis
        - appendicitis
        - cholecystitis
        - cholangitis
        - acute hepatitis
        - inflammatory bowel disease
        - intussusception
        - tumour
        - strangulated hernia
        - volvulus
        - mesenteric ischaemia
        - oesophageal disorders (e.g. achalasia)
        - functional disorders
            - psychogenic
            - irritable bowel
        - pyloric stenosis
    - neurologic
        - head injury
        - CVA
        - idiopathic intracranial hypertension
        - hydrocephalus
        - mass lesion
        - meningitis
        - migraine
        - labyrinthitis
        - Ménière's disease
        - motion sickness
        - cannabinoid hyperemesis syndrome
    - infectious
        - bacterial toxins

        - pneumonia
        - SBP
        - UTI
        - viruses (adenovirus, norovirus, rotavirus)
    - drugs/toxins
        - toxic doses
            - digoxin toxicity
            - aspirin toxicity
            - paracetamol toxicity
            - ipecac toxicity
        - NSAIDs
        - opioids
        - alcohol
        - anticonvulsants
        - antibiotics
        - antiarrhythmics
        - toxins
            - organophosphates
            - carbon monoxide
            - ricin
        - acute radiation syndrome
    - endocrine
        - pregnancy, hyperemesis gravidarum
        - hyponatraemia
        - adrenal insufficiency
        - DKA
        - thyroid/parathyroid disorders
        - uraemia
    - miscellaneous
        - ACS
        - ovarian/testicular torsion
        - nephrolithiasis
        - pain
        - acute angle-closure glaucoma
        - anorexia nervosa/bulimia nervosa
        - depression
- evaluation may include
    - FBC
    - U&E
    - LFT
    - amylase
    - drug levels
    - urine beta hCG
    - urinalysis
    - AXR
    - US
    - CT
- management
    - treat underlying pathology
    - address electrolyte derangements and dehydration as needed
    - antiemetics

    - inhaled isopropyl alcohol (sniffing alcohol wipes)
- complications
    - hypovolaemia
    - metabolic alkalosis
    - hypokalaemia
    - Mallory Weiss tear
    - Boerhaave's syndrome
    - aspiration

GP10 dysphagia

- background
    - defined as difficulty in swallowing
    - usually associated with pharyngeal or oesophageal disease
- symptoms
    - feeling of food sticking
    - discomfort to severe pain
        - patient can usually accurately locate the obstruction
    - regurgitation
    - vomiting
    - choking
    - coughing
    - steady worsening of dysphagia over a few weeks in an older patient suggests malignancy
- aetiology
    - obstructive
        - gastro-oesophageal reflux +/- stricture
            - stricture is usually associated with long history of heartburn
            - one of the most common causes
        - eosinophilic oesophagitis
        - other oesophagitis (e.g. infection)
        - oesophageal cancer
        - gastric cancer
        - pharyngeal cancer
        - post-cricoid web (Paterson-Brown-Kelly syndrome)
        - oesophageal rings
        - foreign body (acute)
        - external pressure from large goitre with retrosternal extension/mediastinal or bronchial tumours
    - neurological
        - cerebrovascular event or brain injury
        - achalasia
        - diffuse oesophageal spasm
        - syringomyelia or bulbar palsy
        - myasthenia gravis
        - multiple sclerosis
        - motor neurone disease
        - myopathy (dermatomyositis, myotonic dystrophy)
        - Parkinson's disease and other degenerative disorders

        - Chagas' disease
    - others
        - pharyngeal pouch
        - globus hystericus
        - external compression
            - e.g. mediastinal tumour or associated with cervical spondylosis
        - CREST syndrome (calcinosis, Raynaud's disease, (o)esophageal dysmotility, sclerodactyly, telangiectasia) or scleroderma
        - oesophageal amyloidosis
        - inflammation (e.g. tonsillitis, laryngitis)
- investigations
    - FBC and ESR/CRP
    - barium swallow and/or endoscopy likely to be required
    - consider laryngoscopic examination if pharyngeal involvement suspected
    - MRI/CT as indicated
    - videofluoroscopy likely to be required for 'difficulty swallowing' rather than 'food sticking', or fibreoptic endoscopic evaluation of swallowing
- management
    - general
        - patient may need to chew well or liquidise food
        - patients with neurological problems may benefit from early Speech and Language involvement
        - eosinophilic oesophagitis may be treated with dietary modification, topical steroids, leukotriene antagonists and other drugs, and endoscopic dilation
    - surgical
        - depending on cause
        - strictures
            - endoscopic dilation with bougies or inflatable balloons
        - oesophageal carcinoma
            - may be suitable for curative surgery (e.g. oesophagectomy) and chemotherapy depending on stage
        - palliative relief of dysphagia in oesophageal carcinoma:
            - repeated dilation
            - stent replacement
            - laser photocoagulation
            - injection of sclerosants
            - brachytherapy can be a useful alternative or adjunct
        - surgical myotomy and endoscopic injection of the sphincter with botulinum toxin are occasionally used for some aetiologies
- complications
    - malnutrition
        - often requires nutritional support prior to treatment
    - aspiration pneumonia
    - iatrogenic perforation
- achalasia
    - background
        - disorder of motility of the lower oesophageal or cardiac sphincter

        - smooth muscle of the oesophagus has impaired peristalsis and failure of the sphincter to relax causes a functional stenosis or functional oesophageal stricture
        - most have no underlying cause but a small proportion are secondary to other conditions such as oesophageal cancer
        - tone and activity of muscle are controlled by a balance of excitatory transmitters such as acetylcholine and substance P and inhibitory transmitters such as nitric oxide and vasoactive intestinal peptide (VIP)
        - local obstruction with proximal dilation is similar to Hirschsprung's disease and often has an aganglionic segment but is apparently acquired rather than congenital and presents much later
        - tends to present in adult life, with a mean age of diagnosis of around 50
            - incidence rare but rising in children
    - presentation
        - most common feature is dysphagia
            - affects solids more than soft foods/liquids
        - food bolus impaction
        - regurgitation in 80-90%
            - some patients learn to induce it to relieve pain
        - chest pain in 25-50%
            - occurs after eating and described as retrosternal
            - more prevalent in early disease
        - heartburn is common and may be aggravated by treatment
        - loss of weight suggests malignancy (which may co-exist)
        - nocturnal cough and sometimes inhalation or refluxed contents is a feature of late disease
        - unlikely to be any findings on examination
    - investigations
        - CXR
            - may show signs of inhalation
            - classical picture shows a vastly dilated oesophagus behind the heart, but rarely seen in practice
            - gastric air bubble may be small or absent
        - barium swallow
            - usually precedes endoscopy to reduce risk of perforating malignancy with endoscope
            - characteristic barium swallow in achalasia:
                - dilated oesophagus with contrast material passing slowly into the stomach as the sphincter opens intermittently
                - distal oesophagus has a narrow segment which resembles a bird's beak on the image
    - endoscopy
        - can detect around a third of achalasia cases
    - manometry of the oesophagus
        - gold standard for diagnosis of achalasia, can detect up to 90% of cases
        - diagnostic features include high resting pressure in the cardiac sphincter, incomplete relaxation on swallowing and absent peristalsis

- lower oesophageal pH monitoring
    - may also be required to exclude GORD
- management
    - calcium channel blockers and nitrates may reduce pressure in the lower oesophageal sphincter but are effective in only around 10%
        - tend to be reserved for patients unable to tolerate other forms of treatment
    - Heller myotomy for those who are fit
        - laparoscopic
        - longitudinal division of the muscle fibres of the lower oesophagus
        - 85-95% success rate with few complications
    - pneumatic dilatation for older unfit patients
        - dilation of a balloon in the lower oesophagus to rupture the muscle whilst leaving the mucosa intact
        - success rate 40-85% with a 0-10% rate of perforation requiring emergency surgery
    - endoscopic injection of botulinum toxin into the lower oesophageal sphincter
        - effective in 85% of cases but benefits reduce over time
- complications
    - untreated achalasia may lead to nocturnal inhalation of material in the oesophagus and aspiration pneumonia
    - treatment may cause oesophageal perforation
    - treatment may lead to GORD
    - longstanding disease increases the risk of oesophageal cancer (possibly because potential carcinogens are held in the oesophagus rather than being moved through)

GC1 alcohol related liver disease including withdrawal

- alcoholic liver disease
    - definition
        - clinical and histological spectrum of disease including fatty liver at one end and alcoholic cirrhosis at the other
        - fatty liver is generally benign and asymptomatic and occurs in patients who abuse alcohol for a period of days to weeks
            - entirely reversible with abstinence from alcohol
            - can develop into alcoholic hepatitis and/or alcoholic cirrhosis
    - pathophysiology
        - development is multifactorial including:
            - female sex
            - viral hepatitis
            - genetics
            - age
            - induction of liver enzymes by other drugs
        - there are various mechanisms of liver damage by alcohol
            - acetate – altered carbohydrate metabolism

        - metabolism of ethanol occurs in the mitochondria where it is oxidised to alcohol dehydrogenase
        - this is oxidised to acetate by acetaldehyde dehydrogenase
        - the reactions alter the redox state of the cell and can have detrimental effects on lipid and carbohydrate delivery, metabolism and export from the liver causing triglyceride accumulation (fatty liver)
    - acetaldehyde – direct hepatocyte damage
        - acetaldehyde concentration is increased in the liver with sustained alcohol intake, resulting in hepatocytes becoming susceptible to hypoxia and hypoxic injury
        - oxygen free radicals cause injury to hepatocytes by lipid peroxidation
    - inflammatory response
        - acetaldehyde causes covalent bonds with proteins which are antigenic
        - long-term exposure to alcohol causes the body to amass circulating antibodies to the proteins resulting in harmful humeral and cellular responses
        - in alcoholic liver injury the expression of pro-inflammatory cytokines us up-regulated, resulting in fibrosis
            - collagen is deposited, progressing to fibrosis and cell linkage formation, resulting in cirrhosis
        - lesions occur in the hepatic veins, causing thickened veins and perisinusoidal fibrosis, which can lead to cirrhosis
    - genetics
        - relationships have been found between alcoholic liver disease and polymorphisms of alcohol metabolising enzyme systems (ALDH2, cytochrome p450) and cytokines (TNK, IL1, IL10)
    - hepatitis C
        - thought to have an additive effective with alcohol
        - the alcohol may alter the ability of the immune system to clear the virus or it may be due to increased iron deposition in liver secondary to high levels of alcohol, which can alter the pathophysiology of the virus
        - hepatitis C and alcoholic liver disease significantly increase the risk of hepatocellular carcinoma
- clinical features
    - alcoholic hepatitis presents with non-specific systemic symptoms and can cause features of portal hypertension
    - alcoholic cirrhosis results in severe liver injury and shrinkage with increased portal hypertension and associated complications as well as decline of liver synthetic function
    - significant damage can occur with no symptoms or signs and the majority are diagnosed on routine blood tests
    - sequalae of alcohol abuse may be the presenting complaint, such as injuries
- clinical history

        - alcohol intake should be part of every clinical history
        - collateral history may also be important
    - spectrum of alcoholic liver disease
        - asymptomatic
            - diagnosed incidentally on routine blood tests
            - may have hepatomegaly on examination
        - early symptoms
            - generally non-specific
                - abdominal discomfort
                - vomiting
                - anxiety
        - injuries associated with intoxication
            - such as falls, rib fractures, head injury, domestic violence
        - alcoholic hepatitis
            - often look more unwell with pyrexia and anorexia
            - LTFs are deranged and they may have clinical signs of chronic liver damage
        - portal hypertension
            - clinical findings are caused by blood being forced down different channels as the portal system pressure rises due to liver damage
            - signs include ascites, varices (rectal and oesophageal), dilated abdominal veins, caput medusae, with the risk of developing encephalopathy
        - cirrhosis
            - end point of liver damage, live tissue is replaced by scar tissue due to sustained damage caused by alcohol
            - synthetic liver function is compromised leading to bleeding and metabolic complications together with the complications of portal hypertension
    - alcohol units
        - one unit
            - half a pint of ‘regular’ beer, lager or cider
            - half a small glass of wine
            - 1 single measure of spirits
            - 1 small glass of sherry
            - 1 single measure of aperitifs
        - more than a unit
            - a pint of ‘regular’ beer, lager or cider
                - =2 units
            - a pint of ‘strong’ or ‘premium’ beer, lager or cider
                - =3 units
            - alcopop or a 275ml bottle of regular lager
                - =1.5 units
            - 440ml can of ‘regular’ lager or cider
                - =2 units
            - 440ml can of ‘super strength’ lager
                - =4 units

- 250ml glass of wine (12%)
    - =3 units
- 75cl bottle of wine (12%)
    - =9 units

- screening tools
    - CAGE questionnaire
        - have you ever felt the need to cut down on your drinking?
        - have you ever felt annoyed by criticism of your drinking?
        - have you ever felt guilty about your drinking?
        - have you ever had a drink (eye opener) first thing in the morning?
    - FAST (fast alcohol screening tool) – a concise version of the AUDIT screening tool
        - how often have you had six or more units if female or eight or more if male, on a single occasion in the last year?
            - never – 0
            - less than monthly – 1
            - monthly – 2
            - weekly – 3 (stop here)
            - daily or almost daily – 4 (stop here)
        - how often during the last year have you failed to do what was normally expected from you because of your drinking?
            - never – 0
            - less than monthly – 1
            - monthly – 2
            - weekly – 3
            - daily or almost daily – 4
        - how often during the last year have you been unable to remember what happened the night before because you had been drinking?
            - never – 0
            - less than monthly – 1
            - monthly – 2
            - weekly – 3
            - daily or almost daily – 4
        - has a relative or friend, doctor, or other health worker been concerned about your drinking or suggested that you cut down?
            - no – 0
            - yes, but not in the last year – 2
            - yes, during the last year – 4
        - an overall score of 3 or more on the first question or all four questions is positive and the remaining AUDIT screening questions should be asked
            - further questions include:
                - how often do you have a drink containing alcohol?
                - how many units of alcohol do you drink on a typical day when you are drinking?

                - how often during the last year have you found that you were not able to stop drinking once you started?
                - how often during the last year have you needed an alcoholic drink in the morning to get yourself going after a heavy drinking session?
                - how often during the last year have you had a feeling of guilt or remorse after drinking?
                - have you or somebody else been injured as a result of your drinking?
            - it groups people into low risk (0-7), increasing risk (8-15), higher risk (16-19) and possible dependence (20 or more)
        - Paddington Alcohol Test
            - starts by identifying whether the patient has one of the top 10 specific triggers:
                - fall (including trip)
                - collapse (including fits)
                - head injury (including facial)
                - assault
                - accident (including burn, RTA)
                - unwell (including requesting detox/help, self-neglect)
                - non-specific GI
                - psychiatric
                - cardiac (including chest pain)
                - repeat attender
            - after dealing with the reason for presentation, they are asked if they drink alcohol and the most they would drink in any one day
                - if more than 8 units, they are asked how often it happens (PAT+ if once a week or more) and whether they feel their current attendance is related to alcohol
            - they can then be offered a brief intervention from an alcohol specialist nurse
- management in the ED
    - the essential component is recognising problem drinkers by including alcohol history in all assessments
    - a brief 10 minute opportunistic advice conversation with those who have hazardous drinking (5 units/day for men, 3 units/day for women) has been found to be beneficial in raising awareness and reducing intake
- symptoms and signs
    - hepatomegaly
        - may be tender in alcoholic hepatitis
    - jaundice
    - cutaneous signs of liver damage
    - ascites
    - encephalopathy
    - bleeding from gastric erosions, exacerbated by coagulopathy
        - more rarely from varices

        - features of alcoholic cirrhosis include:
            - spider naevi
            - enlarged parotid glands
            - gynaecomastia
            - ascites
            - splenomegaly
            - palmar erythema
            - peripheral neuropathy
            - testicular atrophy
    - investigations
        - aspartate aminotransferase (AST) and alanine aminotransferase (ALT)
            - AST/ALT ratio >2 differentiates between alcoholic liver disease and other liver pathology
            - impaired ALT rise results from a hepatic deficiency of pyridoxal-6-phosphate required for ALT enzyme activity in the liver
        - gamma-glutamyltransferase
            - GGT is often raised
            - assesses alcohol abuse indirectly by testing liver damage and has a low sensitivity and specificity of <70%
        - mean corpuscular volume
            - MCV is raised in most cases due to toxic effects of alcohol on bone marrow
            - low sensitivity of around 50%
        - prolongation of prothrombin time
            - PT prolongation and low albumin levels suggest poor synthetic liver function
        - emerging tests:
            - carbohydrate-deficient transferrin test
                - shows the desialylation of transferrin which occurs in the presence of high alcohol intake independent of liver damage
                - thought to be specific for alcoholic liver disease, but has a relatively low sensitivity
            - mitochondrial aminotransferase
                - released from hepatocytes with sustained alcohol abuse
        - ultrasound
            - may be helpful to investigate liver damage
            - fatty liver and alcoholic hepatitis are demonstrated by changes in liver parenchymal reflectivity
            - a cirrhotic liver is irregular and shrunken
            - Doppler studies may show slow or reversed portal vein flow, which, with splenomegaly and intra-abdominal varices, would indicate portal hypertension
        - liver biopsy
            - can be useful to confirm diagnosis prior to treatment
            - with alcoholic liver disease and viral hepatitis, prognostic and management information can be gained from studying necroinflammatory changes at biopsy

        - coagulopathy increases risk of complications and the transjugular venous route is sometimes preferred
- management
    - thiamine deficiency
        - can cause Wernicke's encephalopathy
            - often occurs in chronic alcohol users, particularly during withdrawal
            - characterised by confusion, ataxia, hypothermia, hypotension, nystagmus and vomiting
            - classic triad of confusion, ataxia and opthalmoplegia is only present in around 10% so a high index of suspicion is required
            - if untreated, can progress to Korsakoff's psychosis
                - severe short term memory loss and functional impairment with a requirement for permanent institutionalised care
        - thiamine administration
            - should be given prior to a glucose load to prevent diminished thiamine stores from becoming exhausted and triggering onset of Wernicke's
            - oral thiamine is poorly absorbed in dependent drinkers – parenteral thiamine should be considered in all patients and definitely if any features of Wernicke's are present
            - oral thiamine should be in divided doses to maximise absorption (300mg/day)
    - other management
        - patients may require referral to a GI team for ongoing management
            - those with acute or advanced disease may require admission
        - most patients who are withdrawing can be managed at home
            - those at high risk of complicated withdrawal may need a short inpatient stay
        - observational studies have shown a strong correlation between involvement with Alcoholics Anonymous and long-term abstinence
        - patients requesting detoxification should be advised to make an appointment with their GP and given contact information for community support organisations
- management of alcoholic liver disease
    - the essential part is achieving abstinence from alcohol
        - continuing to drink is the best predictor of a poor outcome
    - diet
        - malnutrition should be improved with a high protein diet
        - patients with at least 2500 kcal/day had improvements in liver function and six month survival compared to those who had less
        - weight loss in obese patients has not been shown to be of benefit in alcoholic fatty liver disease and may worsen liver injury
    - complications of portal hypertension

- possible complications should be considered and managed, such as:
    - encephalopathy
        - differentials include subdural haematoma, Wernicke's encephalopathy and delirium tremens
    - oesophageal varices
    - ascites and liver synthetic function derangement
    - hypoglycaemia
    - coagulopathy
- the only curative treatment for advanced alcoholic liver disease is transplantation
    - current or recent substance abuse is a contraindication
    - selection requires a multi-disciplinary team including a hepatologist, surgeon, addiction specialist, psychiatrist and social worker
    - patients must be motivated and compliant with immunosuppressive treatment
    - relapse of alcoholism after transplant is common
        - the best predictor for continued sobriety is a period of 6 months or longer of documented abstinence
    - alcohol interferes with compliance and metabolism of immunosuppressive medications leading to rapid liver damage in the graft, including cirrhosis

- acute alcohol withdrawal
    - background
        - an estimated 8-11% of patients with alcohol use disorder who are admitted to hospital develop alcohol withdrawal syndrome
        - it is a spectrum of disease defined by DSM-5 as two or more of:
            - insomnia
            - autonomic dysfunction (tachycardia and sweating)
            - tremor
            - nausea and vomiting
            - agitation
            - anxiety
            - seizures (generalised)
            - hallucinations
        - symptoms can develop at any time from a few hours to a few days following cessation of alcohol intake
        - symptoms can range from mild tremor and anxiety to delirium tremens
    - pathophysiology
        - withdrawal develops due to an imbalance in neurotransmitters in patients' brains caused by chronic consumption of ethanol
        - the two key neurotransmitters involved are GABA (inhibitory) and NDMA (excitatory)
        - ethanol potentiates the inhibitory effects of GABA
            - to maintain homeostasis, neurones downregulate GABA and upregulate NDMA
        - on cessation of drinking an imbalance occurs leading to hyperexcitability with loss of the inhibitory balance

        - only a drop in ethanol levels is required, so patients can show symptoms despite having a detectable blood alcohol level
    - screening
        - early identification of at risk patients is important and NICE and RCEM recommend patient screening using questionnaire style assessment tools such as AUDIT or FAST
            - these identify alcohol use disorder and do not predict severity of withdrawal
        - the Glasgow Modified Alcohol Withdrawal Scale (GMAWS) protocol classifies patients with alcohol use disorder plus at least one of the following at high risk of complicated withdrawal:
            - high FAST score (>12) at presentation
            - previous severe withdrawal or alcohol related seizure
            - severe symptoms of withdrawal at presentation
    - requirement for admission
        - NICE recommends admission for medically assisted withdrawal for patients:
            - presenting with or at risk of delirium tremens
            - presenting with or at risk of seizure
            - frail, vulnerable or with multiple co-morbidities
    - investigations
        - basic blood tests and ECG
        - imaging as required
            - alcohol intoxication is an independent risk factor for a positive CT following minor head injury so threshold for scanning should be low
                - the more intoxicated the patient the greater the likelihood of injury
    - management
        - symptom-triggered scoring systems
            - NICE recommends a symptom triggered treatment regime in the acute setting
            - scoring systems assess the severity of the symptoms and guide dosage/frequency of medication
            - benzodiazepines are used in the UK – these potentiate the activity of GABA to restore the balance between GABA and NDMA
            - examples of scoring systems are CIWA-Ar and GMAWS
                - in essence, the more severe the symptoms the larger the dose of benzodiazepines and the more frequent the assessment
                - typical doses would be 10-20mg of diazepam or 1-2mg lorazepam with repeat scoring in 1-2 hours
        - pharmacological therapy
            - NICE does not offer guidelines on special groups; GMAWS suggests:
                - use of lorazepam in patients with alcoholic liver disease due to shorter half life and potential reduced risk of oversedation
                - reduced benzodiazepine doses in patients with co-morbidities (e.g. COPD, reduced GCS, CVD, pneumonia, age

>70, head injury and pregnancy) – dose should be reduced by 50%
        - in patients unable to take oral medication an IV benzodiazepine should be used at 50% of the oral dose
            - this should be delivered by experienced staff and with appropriate monitoring for IV sedation
    - equivalent doses are diazepam 10mg = lorazepam 1mg = chlordiazepoxide 25mg
    - generally speaking diazepam and chlordiazepoxide are thought of as long acting and lorazepam short acting
- severe withdrawal/delirium tremens
    - a small subset of patients develop severe symptoms
    - they can be violent and difficult to manage
    - NICE and GMAWS recommend parenteral benzodiazepines followed by parenteral haloperidol as a second line treatment
        - should only be delivered by clinicians experienced in IV sedation
        - the patients are at high risk of over sedation and complications and will require close observation and likely one to one nursing care
    - other possible treatments (not in widespread use in the UK) for benzodiazepine refractory withdrawal include phenobarbital, dexometamadine and ketamine
    - a small subset of patients require IV propofol infusions and mechanical ventilation due to severity of symptoms

- other complications
    - Wernicke-Korsakoff's syndrome
        - neurological complication caused by nutritional thiamine deficiency
        - Wernicke's presents with confusion, ataxia, nystagmus and ophthalmoplegia
        - Korsakoff's is a progression to cerebellar ataxia, peripheral neuropathy and memory loss
            - patients are classically described as exhibiting confabulation where they fabricate stories to fill in the gaps in their memories
        - NICE recommends that patients with malnourishment or decompensated liver disease presenting with an acute illness should be offered parenteral thiamine replacement
    - refeeding syndrome
        - poor nutritional intake leads to changes in energy metabolism with the body sacrificing muscle stores of glycogen to maintain glucose levels
        - causes depletion of potassium, magnesium, phosphate
        - when the patient is fed again (e.g. on admission to hospital) the adaptations are rapidly reversed, primarily by an increase in insulin levels
        - causes a large shift of extracellular potassium, magnesium and phosphate into cells, causing refeeding, which is characterised by:
            - arrhythmia
            - acute heart failure

                - neurological disturbance – seizure, paraesthesia, muscle weakness, confusion
                - respiratory failure
                - renal failure secondary to acute tubular necrosis
            - NICE recommends that all patients at risk of refeeding should have electrolyte levels (U&E, bone profile, Mg) regularly checked and be referred for review by a dietician
                - electrolyte abnormalities should be promptly corrected
    - hepatic encephalopathy
        - can occur in chronic and acute liver failure
        - caused by the inability of the damaged liver to clear digested proteins (particularly ammonia) which are absorbed by the gut
        - increased serum ammonia causes direct inhibitory effects on neurones via action on GABA and also causes brain oedema
            - this causes initial drowsiness with associated flapping tremor progressing to altered consciousness and coma
            - patients with severe encephalopathy can develop focal neurology such as pupillary signs and extensor posturing
        - diagnosis is clinical
            - imaging may be done (CT/MRI can reveal oedema)
            - EEG may be done
        - serum ammonia can be measured but is not diagnostic
        - management:
            - lactulose is first line for acute and chronic liver disease
            - the antibiotic rifaximin can be added for patients with chronic liver disease and recurrent hepatic encephalopathy
            - both work to alter the gut microbiome to reduce the amount of ammonia-producing bacteria
            - underlying precipitants such as GI bleed and infection should be identified and corrected

GC2 decompensated cirrhosis

- background
    - cirrhosis is a diffuse hepatic process characterised by fibrosis and the conversion of normal liver architecture into structurally abnormal nodules
    - represents the final histological pathway for a variety of liver diseases
    - progression is variable and can be over many weeks or years
    - around 80-90% of the liver parenchyma needs to be destroyed before there are clinical signs of liver failure
    - there is often poor correlation between histological findings and the clinical picture
    - fibrosis causes distortion of the hepatic vasculature which can lead to increased intrahepatic resistance and portal hypertension
        - portal hypertension can lead to oesophageal varices, hypoperfusion of the kidneys, water and salt retention and increased cardiac output
    - damage to hepatocytes causes impaired synthetic function and a reduced ability to detoxify other substances
- causes of cirrhosis

    - common causes
        - alcohol abuse
        - hepatitis B infection
        - hepatitis C infection (up to 20% can develop cirrhosis)
        - non-alcoholic fatty liver disease (NAFLD) and non-alcoholic steatohepatitis (NASH) – up to 10% of patients with NASH can develop cirrhosis
    - less common causes include:
        - haemochromatosis
        - primary biliary cirrhosis
        - biliary obstruction
            - e.g. due to biliary atresia/neonatal hepatitis, congenital biliary cysts, cystic fibrosis
        - autoimmune hepatitis
        - inherited metabolic disorders e.g.:
            - tyrosinaemia
            - Wilson's disease
            - porphyria
            - alpha-1-antitrypsin deficiency
            - glycogen storage disease
        - sarcoidosis or other granulomatous disease
        - primary sclerosing cholangitis
        - venous outflow obstruction in Budd-Chiari syndrome or veno-occlusive disease
        - drugs and toxins including methotrexate, amiodarone, isoniazid
        - congestive heart failure or tricuspid regurgitation
            - rarely seen now due to improved management
        - infections including congenital and tertiary syphilis and schistosomiasis
- epidemiology
    - there are an estimated 30,000 people in the UK with diagnosed cirrhosis and at least 7000 new diagnoses per year
    - there are concerns that increasing alcohol consumption at dangerous levels may lead to ab increase
- risk factors for cirrhosis
    - there may a genetic disposition which would explain different rates of development in patients with similar risk factors
    - chronic alcohol consumption increases the risk of progression of cirrhosis from any cause
    - risk factors for the development of cirrhosis in patients with chronic hepatitis C infection include:
        - regular (moderate) alcohol consumption
        - age >50
        - male sex
    - risk factors for the development of cirrhosis in patients with NASH include:
        - older age
        - obesity
        - insulin resistance or type 2 diabetes
        - hypertension

        - hyperlipidaemia
- presentation
    - commonly asymptomatic until obvious complications occur
    - up to 40% of patients with cirrhosis may be asymptomatic
    - history should include any possible underlying causes of cirrhosis including full drug and alcohol history (including OTC, complimentary and recreational drugs), risk factors for hepatitis infection, family history of autoimmune or liver diseases
    - symptoms
        - initial symptoms of cirrhosis may be vague, e.g.:
            - fatigue
            - malaise
            - anorexia
            - nausea
            - weight loss
        - in advanced decompensated liver disease:
            - oedema
            - ascites
            - easy bruising
            - poor concentration and memory
            - bleeding oesophageal varices
            - spontaneous bacterial peritonitis
    - signs
        - physical signs are variable and depend upon the extent of the disease
        - cutaneous features of cirrhosis include:
            - jaundice
            - scratch marks secondary to pruritus
            - spider naevi (mainly on trunk and face)
            - skin telangiectasias ('paper money skin')
            - palmar erythema
            - bruising
            - petechiae or purpura
            - hair loss
            - white nails (hypoalbuminaemia)
            - finger clubbing
            - Dupuytren's contracture
        - other signs:
            - hepatomegaly and a nodular liver
            - oedema
            - gynaecomastia and male hair loss pattern
            - hypogonadism/testicular atrophy/amenorrhoea
                - due to the direct toxic effect of alcohol or iron in haemochromatosis
            - Kayser-Fleischer ring (pathognomic for Wilson's disease)
        - signs of portal hypertension
            - ascites
                - clinically detectable when more than 1.5 litres of fluid present

        - caput medusae (veins radiating from the umbilicus)
        - enlarged spleen
    - signs of hepatic encephalopathy
        - asterixis
            - demonstrated by hyperextending the wrist, pushing gently on the joints of the four fingers
            - a slow clonic flexion-relaxation response occurs after a few seconds

- investigations
    - bloods
        - LFTs
            - including AST, ALT, ALP, bilirubin, gamma-GT
            - AST and ALT are raised due to hepatocyte damage
            - gamma-GT is high in active alcoholics
        - albumin
            - hypoalbuminaemia in advanced cirrhosis
        - FBC
            - anaemia from occult bleeding
            - thrombocytopaenia from hypersplenism
            - macrocytosis
        - renal function tests
            - hyponatraemia due to increased ADH activity
            - poor renal function in hepatorenal syndrome
        - red cell folate
            - diet may be folate inadequate
        - coagulation screen
            - sensitive test of liver function
        - ferritin
            - low levels may indicate iron deficiency from diet or blood loss
            - raised in haemochromatosis
        - viral antibody screen
            - to look for evidence of hepatitis B or C infection
        - fasting glucose/insulin/triglycerides and uric acid levels
            - if NASH suspected
        - autoantibody screen
            - anti-mitochondrial antibodies in primary biliary cirrhosis
        - alpha-1-antitrypsin levels
        - ceruloplasmin and urinary copper if Wilson's disease suspected
        - fasting transferrin saturation and haemochromatosis C282Y if haemochromatosis suspected
    - imaging
        - ultrasound (+/- CT or MRI) of liver
            - mainly to detect complications such as splenomegaly, ascites, hepatocellular carcinoma
        - CXR
            - may show elevated diaphragm +/- pleural effusion (passage of ascitic fluid across diaphragm)

        - specialist imaging and biopsy may be required
- classification of cirrhosis
    - Child-Pugh-Turcotte classification
        - serum albumin (g/L)
            - >35 (1)
            - 28-35 (2)
            - <28 (3)
        - serum bilirubin (total)
            - <34 μmol/L (1)
            - 34-50 μmol/L (2)
            - >50 μmol/L (3)
        - INR
            - <1.7 (1)
            - 1.7-2.2 (2)
            - >2.2 (3)
        - ascites
            - absent (1)
            - controlled medically (2)
            - poorly controlled (3)
        - encephalopathy
            - absent (1)
            - controlled medically (2)
            - poorly controlled (3)
        - scoring
            - 5-6 = class A (life expectancy 15-20 years)
            - 7-9 = class B (life expectancy 4-14 years)
            - 10-15 = class C (life expectancy 1-3 years)
- management
    - aim is to delay progression of cirrhosis and to prevent and/or treat any complications
    - specific treatment for underlying cause
    - adequate nutrition, including protein and calorie intake
    - alcohol
        - most important measure for someone with alcoholic cirrhosis
        - alcohol also increase rate of progression from any source
    - zinc supplements (zinc deficiency is common)
    - management of pruritus
        - common in both cholestatic and non-cholestatic liver disease
        - mild itch may respond to antihistamines and topical ammonium lactate
        - colestyramine
        - rifampicin has helped some patients unresponsive to colestyramine
        - severe pruritus may require treatment with UV light or plasmapheresis
    - those at risk of osteoporosis should be given preventative treatment
    - regular exercise to prevent muscle wasting
    - prophylactic antibiotic use in patients with cirrhosis and upper GI bleeding significantly reduces bacterial infections and seems to reduce all-cause mortality, bacterial infection mortality, rebleeding events and length of hospitalisation

- patients with chronic liver disease should be vaccinated against hepatitis A, influenza and pneumococci
- caution with prescribing of drugs that may not be properly metabolised in the presence of liver failure
- liver transplantation is the ultimate treatment

- complications
  - if complications develop, the person should be transferred to a specialist liver unit where there is expertise to manage the complications and assess suitability for liver transplant
  - anaemia, thrombocytopenia and coagulopathy
    - anaemia may result from folate deficiency, haemolysis or hypersplenism
    - thrombocytopenia is usually secondary to hypersplenism and decreased levels of thrombopoetin
    - coagulopathy results from decreased hepatic production of coagulation factors
      - cholestasis, if present, causes decreased vitamin K absorption, leading to reduced hepatic production of factors II, VII, IX and X
    - patients with cirrhosis may also develop fibrinolysis and DIC
  - oesophageal varices
    - as a result of portal hypertension
    - young people and adults with cirrhosis and upper GI bleeding should be given prophylactic antibiotics at presentation
    - endoscopic variceal band ligation should be offered for primary prevention of bleeding for people with medium to large oesophageal varices
    - patients with a new diagnosis of cirrhosis should be offered upper gastrointestinal endoscopy
      - surveillance should be done every 3 years for those without varices on the first scope
  - ascites
    - accumulation of excessive fluid in the peritoneal cavity due to increased plasma volume 'spilling over' into the abdominal cavity
    - ultrasound can help to detect smaller volumes
    - patients with refractory ascites may require a transjugular intrahepatic portosystemic shunt
    - prophylactic oral ciprofloxacin or norfloxacin should be offered to people with cirrhosis and a an ascitic protein of 15g/L or less until the ascites has resolved
  - spontaneous bacterial peritonitis
    - thought to be caused by spread of bacteria across the gut wall and/or haematogenous bacterial spread
    - E. coli is one of the most common organisms
    - may be prevented by adequately treating ascites and treating those with high neutrophil counts (>250 neutrophils/ml) in their ascitic fluid with empirical IV antibiotics and albumin
    - patients who survive an episode should receive long term oral antibiotic prophylaxis
  - hepatocellular carcinoma
    - risk varies according to cause of cirrhosis

        - most often associated with cirrhosis caused by hepatitis C infection, followed by hereditary haemochromatosis
        - worldwide, hepatocellular carcinoma secondary to cirrhosis caused by hepatitis B infection causes a large number of deaths
        - risk is lower in people with alcoholic cirrhosis (8% five year occurrence) or primary biliary cirrhosis (4% five year occurrence)
        - patients with cirrhosis should be screened for hepatocellular carcinoma with ultrasound, triphasic CT or MRI
    - other complications
        - less common complications can include:
            - cirrhotic cardiomyopathy
                - cardiac hypertrophy and a blunted stress response of the heart
                - may cause significant perioperative problems and mean that transplantation is too dangerous
            - hepatopulmonary syndrome
                - pulmonary arteriolar vasodilation, shunting and hypoxaemia
                - may be reversed with transplantation
            - portopulmonary hypertension
                - an irreversible condition that can occur in those with ascites
            - surgery and general anaesthesia have increased risks

GC3 dehydration in children

- background
    - clinical assessment of dehydration can be difficult and is rarely accurate
    - most useful signs for predicting 5% dehydration in children are an abnormal capillary refill time, abnormal skin turgor and abnormal respiratory pattern
- clinical assessment
    - body weight
        - normal: no loss of body weight
        - mild dehydration: 5-6% loss of body weight
        - moderate dehydration: 7-10% loss of body weight
        - severe dehydration: >10% loss of body weight
    - clinical features of mild to moderate dehydration – 2 or more of:
        - restlessness or irritability
        - sunken eyes
        - thirsty and drinks eagerly
    - clinical features of severe dehydration – 2 or more of:
        - abnormally sleepy or lethargic
        - sunken eyes
        - drinking poorly or not at all
        - pinch test for skin turgor – unreliable in obese or severely malnourished children
            - normal – skin fold retracts immediately
            - mild or moderate dehydration – skin fold visible for <2 seconds
            - severe dehydration – very slow, skin fold visible for >2 seconds
    - other features of dehydration:

        - dry mucous membrane
        - reduced tears
        - decreased urine output
    - other signs of severe dehydration:
        - circulatory collapse (weak rapid pulse, cool or blue extremities, hypotension)
        - rapid breathing
        - sunken anterior fontanelle
- clinical assessment of hydration status
    - mild/not clinically detectable dehydration (<5%)
        - symptoms
            - appears well
            - alert and responsive
            - normal urine output
            - skin colour unchanged
            - warm extremities
        - signs
            - alert and responsive
            - skin colour unchanged
            - warm extremities
            - eyes not sunken
            - moist mucous membranes
            - normal heart rate
            - normal breathing pattern
            - normal peripheral pulses
            - normal capillary refill time
            - normal skin turgor
            - normal blood pressure
    - clinical dehydration (5-10% loss in body weight)
        - symptoms
            - appears to be unwell or deteriorating
            - altered responsiveness – irritable, lethargic
            - decreased urine output
            - skin colour unchanged
            - warm extremities
        - signs
            - altered responsiveness – irritable, lethargic
            - skin colour unchanged
            - warm extremities
            - sunken eyes
            - dry mucous membranes (except for mouth breathers)
            - tachycardia
            - tachypnoea
            - normal peripheral pulses
            - normal capillary refill time
            - reduced skin turgor
            - normal blood pressure

- clinical shock (>10% loss of body weight)
    - symptoms
        - decreased level of consciousness
        - pale or mottled skin
        - cold extremities
    - signs
        - decreased level of consciousness
        - pale or mottled skin
        - cold extremities
        - tachycardia
        - tachypnoea
        - weak peripheral pulses
        - prolonged capillary refill time
        - hypotension (decompensated shock)

- management
    - no clinical dehydration
        - aim is to prevent dehydration
        - patient can be discharged and managed at home
        - verbal advice:
            - continue breastfeeds and other feeds
            - encourage fluid intake
            - discourage fruit juices and carbonated drinks
            - if increased risk of dehydration, offer low osmolality ORS (Dioralyte) as a supplemental fluid
            - when to seek advice from a healthcare professional:
                - appearing to get more unwell
                - changing responsiveness (e.g. irritability, lethargy)
                - decreased urine output
                - pale or mottled skin
                - cold extremities
            - advice on likely duration of symptoms:
                - vomiting: 1-2 days, most stop within 3 days
                - diarrhoea: 5-7 days, most stop within 2 weeks
    - clinical dehydration
        - oral rehydration
        - continue to breastfeed if applicable
            - otherwise use low osmolality ORS
        - calculate deficit (estimated at 5% in this category)
        - calculate fluid maintenance requirements
        - replace over 4 hours in frequent but small amounts
            - total replacement rate is usually 10-20ml/kg/hr
            - deficit (ml) = 5 x weight x 10
                - add to daily maintenance/24 x 4 for amount over 4 hours
        - monitor response to oral fluids
        - if ORS is refused by the child and there are no red flags, consider other fluids (e.g. milk, water)
            - avoid fruit juices and carbonated drinks

        - consider NG tube placement if the child is unable to drink and/or vomits persistently
            - IV rehydration and/or admission may be required
        - discharge criteria
            - consider discharge home if oral fluid tolerated over first hour
            - reassure parents that oral rehydration is usually possible
            - verbal advice:
                - complete the remainder of the 4 hours fluid challenge at home
                - administer fluid in small, frequent amounts
                - breastfeeding should be continued throughout rehydration
                - age-appropriate diet should be started after initial rehydration
                - seek advice if the child refuses to drink or vomits persistently
    - clinical shock
        - resuscitation phase
            - ensure patent airway and give oxygen
            - obtain urgent IV access
                - U&Es, blood glucose, VBG
            - fluid bolus of 20ml/kg 0.9% sodium chloride
            - if remaining shocked after first bolus:
                - give second bolus
                - consider other causes for shock
            - if remaining shocked after second bolus:
                - give further bolus
                - consider discussion with paediatric intensive care team
        - maintenance phase
            - once symptoms and signs of shock have resolved
            - calculate daily maintenance requirement
            - use 10% estimate for deficit calculation
            - consider adding potassium to fluids once serum level is known
            - monitor clinical and laboratory response to fluid therapy and adjust subsequent fluids as appropriate
            - discuss with paediatric team
- choice of rehydration fluid
    - oral or NG rehydration
        - reduced osmolarity oral rehydration solution (ORS)
            - 50-60mmol/L sodium
            - lemonade, sports drinks and homemade ORS are not appropriate
    - IV rehydration
        - isotonic fluid (usually 0.9% sodium chloride) during the initial phase of resuscitation
        - once volume has been restored, glucose should be added in the maintenance phase
            - 0.9% sodium chloride with 5% dextrose
- volume and rate of fluid replacement

- oral rehydration
    - aim for 10-20ml/kg of ORS in frequent small amounts
    - replace deficit over 4 hours
- NG rehydration
    - two regimes described:
        - rapid NG replacement
            - 25ml/kg/hr of ORS over 4 hours
        - standard NG replacement
            - replace deficit over first 6 hours
            - give maintenance over the next 18 hours
            - the slower regime is preferred in infants <6 months, in the presence of significant co-morbidities or for children with significant abdominal pain
- IV rehydration
    - resuscitation phase
        - IV boluses of 20ml/kg 0.9% sodium chloride
    - standard rehydration regime
        - calculate total deficit and maintenance requirement and replace over 24 hours
    - rapid IV rehydration regime
        - 20ml/kg/hr 0.9% sodium chloride for 2-4 hours followed by oral rehydration
        - WHO recommends that IV rehydration should be completed within 3-6 hours depending on age

- calculating deficit and maintenance
    - deficit
        - estimate if deficit is 5% or 10%
        - estimated deficit = 5 (or 10) x child's weight in kg x 10
    - maintenance
            - 100ml/kg for first 10kg then
            - 50ml/kg for next 10kg then
            - 20ml/kg for each subsequent kg
        - divide by 24 to get hourly maintenance requirements

GC4 functional bowel disorders

- irritable bowel syndrome
    - background
        - relapsing bowel disorder where abdominal pain is associated with defecation or a change in bowel habit
        - defined by symptom-based diagnostic criteria in the absence of detectable organic causes
        - reclassified from a functional disorder to a disorder of brain-gut interaction
        - diagnosis rarely changes over time but may need re-evaluation if symptoms change
    - epidemiology
        - present in 10-20% of the UK population, but prevalence is likely higher as many people do not seek medical attention

        - global prevalence varies widely (1-45%)
        - more common in women
        - peak prevalence is between 20 and 30 years
    - aetiology
        - no structural lesion and no single explanation for the condition
            - seems to involve abnormal smooth muscle activity +/- visceral hypersensitivity and abnormal central processing of painful stimuli
            - bidirectional communication between the gut and the brain is modulated by the autonomic nervous system
                - a reduction in parasympathetic activity and increase in sympathetic activity is seen in many IBS patients
        - associated with increased levels of stress and poor coping strategies
        - 20-60% have enhanced visceral perception to various physiological stimuli
        - can occur in families and there may be a genetic component
        - subclasses
            - 1/3 have IBS with constipation (IBS-C)
                - loose stools <25% and hard stools >25% of the time
            - 1/3 have IBS with diarrhoea (IBS-D)
                - loose stools >25% and hard stools <25% of the time
            - the remainder have IBS mixed (IBS-M)
                - both hard and soft stools >25% of the time
        - colonic transit is abnormal in only 10-20% of patients with IBS-C and IBS-M and 25-45% of patients with IBS-D
            - transit times do not equate to motility
        - immune system modulation may play a role, with increased mast cell activity associated with low grade mucosal inflammation sometimes seen in post-infectious IBS
        - there is good evidence that bacterial, viral or parasitic infections can trigger IBS
            - antibiotics can either improve or worsen the situation
    - presentation
        - the British Gastroenterology guidelines use the Rome IV criteria
            - presence of abdominal pain related to defecation associated with a change in stool frequency and/or stool form
            - frequency of abdominal pain at least one day per week
        - NICE found the criteria too restrictive and suggests the following:
            - at least a six month history of either:
                - abdominal pain or discomfort
                - bloating
                - change in bowel habit
            - consider positive diagnosis only if abdominal pain is either relieved by defecation or associated with altered stool frequency or form and at least two of:
                - altered passage of stool (straining, urgency, incomplete evacuation)
                - abdominal bloating (women >men), distension, tension or hardness

        - symptoms aggravated by eating
        - passage of mucus rectally
    - supporting symptoms include lethargy, nausea, backache and bladder symptoms
    - other features
        - most patients have abdominal pain and disordered bowel habit, continuous or intermittent
        - a 'morning rush' is common – the urgent need to defecate several times on getting up, before and after breakfast
        - symptoms are chronic, with remissions interrupted by relapses precipitated by stress or changes in bowel flora produced by antibiotics
        - upper GI symptoms may include nausea, heartburn, dysphagia and early satiety
        - extra-intestinal symptoms include:
            - headache
            - migraine
            - asthma
            - backache
            - lethargy
            - dyspareunia
            - urinary frequency and urgency
            - psychological problems such as anxiety and depression are also more common but may be associated with healthcare seeking rather than IBS per se
- signs
    - abdominal and digital rectal examination can help exclude other diagnoses
    - dyssynergic defecation (paradoxical contraction on rectal examination during straining) or low rectal masses may be identified
- differentials
    - colon cancer
    - inflammatory bowel disease
    - bile acid malabsorption
    - coeliac disease
    - gastroenteritis
    - diverticular disease
    - gut neuroendocrine tumours such as carcinoid
    - gynaecological problems
        - e.g. pelvic inflammatory disease, endometriosis, ovarian tumours
    - anxiety +/- depression, somatisation, panic disorders
- investigations
    - diagnosis should be made positively on symptom-based criteria, not as a diagnosis of exclusion after ruling out organic disease after extensive investigation
    - any family history of inflammatory bowel disease or colon cancer <50 should lower the threshold for investigation/referral
    - patients meeting the criteria for IBS should have the following:

                - FBC
                - ESR
                - CRP
                - antibody testing for coeliac disease (endomysial antibodies or tissue transglutaminase)
                - CA 125 for women with symptoms which could be ovarian cancer
                - faecal calprotectin for those with symptoms which could be inflammatory bowel disease
            - tests that are NOT required in those meeting the diagnostic criteria include:
                - TFTs
                - ultrasound
                - colonoscopy/sigmoidoscopy/barium enema
                - faecal occult blood
                - faecal ova and parasite tests
                - hydrogen breath tests
    - criteria for further investigation include:
        - diagnostic uncertainty, alarm symptoms, severe resistant symptoms
        - red flag symptoms:
            - unintentional weight loss
            - unexplained rectal bleeding in patients 50 or over
            - family history of bowel or ovarian cancer
            - aged over 60 with a change in bowel habit >6 weeks with looser or more frequent stools
            - anaemia
            - abdominal or rectal masses
            - raised inflammatory markers
        - it is important to avoid unnecessary referral and intervention, and these may prolong anxiety rather than relieve it
    - management
        - reassurance and explanation
        - lifestyle and physical activity
            - information about the condition and self-help
            - encourage patients to identify and make best use of leisure time and create times in the day for relaxation
            - advice on increasing physical activity if appropriate
                - there is evidence that this has a positive effect on symptoms
        - diet
            - general dietary advice
                - regular meals, not rushed and avoiding long gaps between them
                - plenty of fluids with restricted tea and coffee (max 3 cups/day)
                - reduced intake of alcohol and fizzy drinks
                - consider limiting high fibre foods and resistant starches (e.g. from processed/recooked foods or fresh fruit)
                - for diarrhoea – avoid sorbitol

        - for wind – consider increasing oats and linseed (one tablespoon/day)
    - fibre
        - those with constipation may need to increase fibre intake and those with diarrhoea may need to reduce it
        - FODMAPs
            - fermentable oligosaccharides, disaccharides, monosaccharides and polyols
            - foods high in FODMAPs may have fermentation and osmotic effects, increasing symptoms
            - examples include apples. cherries, peaches, artificial sweeteners, most lactose-containing foods, legumes, many green vegetables
            - up to 86% of people may have an improvement in symptoms, specifically bloating, abdominal pain, flatus and altered bowel habit, on a low FODMAP diet
            - concerns include nutritional adequacy, cost and difficulty in teaching and maintaining the diet – most of which can be addressed by a nutritionist
    - dietician
        - referral may be helpful
    - probiotics
        - there is some evidence for multi-strain probiotic supplements being useful but optimal regimes and products are not known
        - NICE suggests taken for four weeks at the recommended dose while monitoring the effect
- pharmacological treatments
    - there is a high rate of placebo effect even when the patient knows they are taking a placebo
    - pharmacological treatments should target the individual symptoms
    - loperamide
        - medication of choice for diarrhoea
    - antispasmodics
        - for abdominal pain and spasms
        - options include alverine, mebeverine and peppermint oil
    - laxatives
        - may be required for constipation
        - lactulose should be avoided
    - antidepressants
        - can be of benefit
        - TCAs and SSRIs can both be effective
        - NICE guidelines advocate use of an SSRI only if a low-dose TCA has not been effective

        - treatment should be started at a low dose (e.g. 10mg amitriptyline) and increased if necessary to no more than 30mg
    - antibiotics
        - variable effect in IBS
        - the non-absorbable antibiotic rifaximin ahs been used as a second line drug for patients with diarrhoea
    - 5-hydroxytryptamine 3 receptor agonists
        - second line drugs for diarrhoea used in secondary care
        - ondansetron may be considered, from 4mg OD to 8mg TDS
- other therapies
    - psychological therapy
        - recommended by NICE
        - includes cognitive behavioural therapy, hypnotherapy and/or psychological therapy
        - considered for patients who do not respond to pharmacological treatments after 12 months
        - NICE guidance advises against acupuncture or reflexology for IBS
- ED management options
    - avoid opiates
    - IV Buscopan
        - can be repeated as necessary
        - response confirms diagnosis of gastrointestinal spasm as it has no analgesic properties
    - oral Buscopan
        - to manage ongoing issues
        - only 10% absorbed orally
            - some patients may need to take more than the proscribed dose, such as 4-6 tablets at once, repeated as needed
        - IM Buscopan self administered may be considered for acute attacks to reduce ED attendances
    - IV paracetamol
        - can be added if needed
    - diazepam
        - may be useful to manage the distress of the intensity of the pain
        - can be administered rectally if there is vomiting
    - GTN
        - some patients have severe chest pain indistinguishable from cardiac pain
            - may radiate to the middle of the back which can be relatively uncommon in angina
        - there is often a good response to GTN
        - cardiac causes need to be ruled out
        - patients may also respond well to diazepam, IV Buscopan and IV paracetamol
        - tricyclic antidepressants can help to reduce the frequency of attacks

- prognosis
    - symptoms fluctuate over many years
    - more than 50% continue to have symptoms after 7 years
    - people with a long history are less likely to improve
    - ongoing stress may hinder recovery
    - IBS is not associated with the long-term development of any disease
        - patients with IBS do have an increased risk of cholecystectomy
    - the post-infective subgroup has a better prognosis with symptoms resolving in many after 5-6 years

- cyclical vomiting syndrome
    - background
        - a condition in which there are repeated episodes of severe nausea, vomiting and physical exhaustion
        - more common in children but can present in adulthood
        - rare
            - thought to affect around 3 in 100,000 children
        - average age of first presentation is 5 years
        - females slightly more affected than males
    - aetiology
        - no known cause
        - more common in those who have migraines
            - 80% of children and 25% of adults with cyclical vomiting syndrome also have migraines
        - there is an overlap between cyclical vomiting syndrome and abdominal migraine
        - more common in people with a family history of migraine
        - pathogenesis is likely to be multifactorial, with multiple genetic, autonomic, central and environmental factors playing a role
    - presentation
        - clinical features resemble those found in association with migraine headaches
        - main symptoms are severe nausea and sudden vomiting
            - can last from a few hours to a few days
        - four phases:
            - prodromal
                - often marked by intense sweating and nausea
                - the person may look pale
                - lasts from a few minutes to several hours
            - vomiting
                - lasts from hours to days
                - nausea, vomiting and retching last for 20-30 minutes at a time
            - recovery
                - cessation of vomiting and retching
                - improving appetite
                - return of energy
            - well

        - symptom-free stage
        - following an episode the patient is symptom free for several week or months
    - other symptoms include:
        - lack of appetite
        - abdominal pain
        - diarrhoea
        - dizziness
        - photophobia
        - headache
    - severity of episodes varies between cases
        - episodes tend to start at the same time of day, last the same length of time and occur with the same symptoms and level of intensity
        - some people may require admission
    - possible triggers for episodes:
        - emotional stress
        - anxiety
        - infections, especially sinusitis
        - certain foods – e.g. chocolate, cheese, monosodium glutamate
        - long periods of time without food
        - dehydration
        - hot weather
        - menstrual periods
        - excess exercise
        - sleep deprivation
- diagnosis
    - in children
        - at least five episodes, or a minimum of three over a six month period
        - episodic attacks of intense nausea and vomiting lasting one hour to ten days, occurring at least one week apart
        - stereotypical pattern and symptoms in the individual patient
        - vomiting during episodes occurring at least four times an hour for at least one hour
        - a return to baseline health between episodes
        - symptoms cannot be attributed to another disorder
    - in adults
        - stereotypical episodes of vomiting regarding onset (acute) and duration (less than one week)
        - a minimum of three discrete episodes in the preceding year
        - absence of nausea and vomiting between episodes
        - no metabolic, gastrointestinal or CNS structural or biochemical disorders
- differential
    - GORD
    - gastritis, duodenitis
    - Crohn's disease

        - ulcerative colitis
        - migraines
        - acute intermittent porphyria
        - Addison's disease
        - DKA
        - phaeochromocytoma
    - investigations
        - diagnosis is usually clinical
        - should be considered in any child who has repeated episodes of vomiting with wellness in between
        - any investigations are usually to exclude any underlying conditions
            - e.g. FBC, U&E, gastroscopy, abdominal ultrasound
        - pregnancy test for older girls and women
    - management
        - avoidance of triggers
            - e.g. certain foods, stress, sleep deprivation, avoiding dehydration
        - prophylactic and abortive therapy
            - preventative medications are considered for patients with more than one episode a month
            - prophylactic treatments include amitriptyline, propranolol and topiramate
            - medications for aborting acute episodes include ondansetron, prochlorperazine and triptans
            - adding erythromycin to standard propranolol treatment has been shown to improve the response in children
        - supportive care during acute episodes
            - e.g. IV fluids, analgesia
        - family support
    - prognosis
        - full recovery in the majority of cases
        - dehydration can occur in more severe or prolonged cases
        - oesophagitis or Mallory-Weiss tear can occur due to the excessive vomiting
        - tooth decay can occur in some cases
        - most cases resolve in late childhood or early adolescence
            - around half of children develop migraines when they are older
        - parents and children have lower health-related quality of life compared to children with irritable bowel syndrome
- abdominal migraine
    - diagnostic criteria (all of the following at least twice in the preceding twelve months):
        - paroxysmal episodes of intense, acute periumbilical pain lasting at least an hour
        - intervening periods of normal health, lasting weeks to months
        - pain interferes with normal activities
        - pain is associated with two or more of:
            - anorexia
            - nausea

                - vomiting
                - headache
                - photophobia
                - pallor
            - after appropriate medical evaluation the symptoms cannot be attributed to another medical condition

GC5 gastrointestinal infections

- gastroenteritis
    - background
        - caused by a variety of viral (e.g. norovirus, rotavirus, adenovirus), bacterial (e.g. Campylobacter spp, Salmonella spp, Shigella spp or toxins from Staph aureus, Bacillus cereus, Clostridium perfringens) or parasitic pathogens (e.g. Cryptosporidium spp, Entamoeba histolytica, Giardia lamblia)
        - estimated 17 million cases in the UK per year
        - viral infections cause 30-40% of cases in industrialised countries
        - the causative agent for most cases is not identified
    - risk factors
        - poor personal hygiene and lack of sanitation
        - compromised immune system
        - achlorhydria including from acid-suppressing drugs
        - poor storage or insufficient reheating of foods
    - presentation
        - incubation period for viruses is around 1 day, for bacillary dysentery a few hours to four days and for parasites 7-10 days
    - assessment
        - check observations
        - thorough abdominal examination to consider other possible diagnoses
        - assess for features of dehydration
            - mild dehydration
                - lassitude
                - anorexia
                - nausea
                - light-headedness
                - postural hypotension
            - moderate dehydration
                - apathy
                - tiredness
                - dizziness
                - muscle cramps
                - dry tongue
                - sunken eyes
                - reduced skin elasticity
                - postural hypotension
                - tachycardia
                - oliguria
            - severe dehydration

        - profound apathy
        - weakness
        - confusion (leading to coma)
        - shock
        - tachycardia
        - marked peripheral vasoconstriction
        - systolic BP <90mmHg
        - oliguria or anuria
- investigations
    - stool for culture/cysts/ova/parasites, culture and sensitivity
        - acute painful diarrhoea or blood, mucus or pus in stool
        - systemically unwell or immunocompromised patient
        - recent antibiotic or PPI or hospital admission
        - suspected food poisoning
        - recent foreign travel
        - recurrent diarrhoea, not improved by day 7 or not resolved by day 14
        - uncertainty of diagnosis of gastroenteritis
        - diarrhoea in a person at risk of transmitting infection (e.g. food handler, clinical worker, child in childcare/school)
    - bloods may be needed for unwell patients (FBC, U&E)
    - other tests depending on clinical picture (e.g. imaging)
- notification
    - dysentery and food poisoning are notifiable
- management
    - patient information leaflets
    - advice on prevention of dehydration
        - healthy adults
            - regular fluid intake +/- fruit juice and soup
        - increased risk of dehydration (e.g. elderly, immunocompromised, comorbid)
            - oral rehydration solution
        - adults with clinical features of dehydration who can be safely managed at home
            - frequent small amounts of ORS (e.g. 200-400ml after every loose motion)
    - antidiarrhoeals
        - not recommended
            - may be considered in mild to moderate diarrhoea to allow someone to resume essential activities
            - contraindicated if:
                - blood, mucus and/or pus in stools and high fever (possible dysentery)
                - shigellosis or confirmed, probable or suspected STEC O157 infection
    - advice on methods of preventing transmission
        - thorough hand washing with liquid soap and warm running water

- use of flush toilet where possible
- avoid sharing of towels/flannels
- soiled clothing and bedlinen should be washed separately at a high temperature (60 degrees or higher)
    - washing machine should not be more than half full

- advice on isolation
    - person should not attend work or other institutional/social settings until at least 48 hours after the last episode of diarrhoea or vomiting
        - some infections may require longer periods as per specialist advice

- consideration of admission
    - vomiting and unable to keep down oral fluids
    - features of sepsis
    - features of severe dehydration
    - inadequate response to oral rehydration solution
    - recent foreign travel
    - older age
    - poor home circumstances and low level of support
    - fever
    - bloody diarrhoea
    - abdominal pain and tenderness
    - faecal incontinence
    - diarrhoea lasting more than 7 days
    - co-existing medical conditions and drug therapies (e.g. systemic steroids, ACEi, diuretics)
- complications
    - dehydration and electrolyte disturbance
    - haemolytic uraemic syndrome
        - rare, characterised by acute kidney injury, haemolytic anaemia and thrombocytopenia
        - most common in young children and the elderly
    - reactive complications
        - e.g. arthritis, carditis, urticaria, erythema nodosum, conjunctivitis, reactive arthritis
    - systemic invasion by Salmonella spp
        - can cause endovascular infections and localised infections in bones, joints, meninges, gallbladder
    - toxic megacolon caused by fulminant colitis
        - rare
    - Guillain-Barré syndrome
        - associated with a number of viruses, most commonly Campylobacter jejuni
    - malnutrition
    - intractable diarrhoea
    - thrombotic thrombocytopenic purpura
    - irritable bowel syndrome
    - lactose intolerance

        - reduced absorption of drugs such as anticonvulsants and contraceptives
- norovirus
    - presentation
        - incubation period usually 24-48 hours but may be as low as 12 hours
        - common symptoms include fever, nausea and vomiting that may be projectile along with watery diarrhoea without blood
        - abdominal cramps are common
        - symptoms last 12 to 60 hours
        - most people make a full recovery in 1-2 days but diarrhoea may last longer
        - seizures occasionally occur, but long term neurological sequelae are uncommon
    - management
        - avoidance or correction of dehydration
        - fluid and electrolyte replacement
        - avoid anti-diarrhoeals and antiemetics
        - increased frequency of cleaning and good handwashing
        - infectivity lasts for 48 hours after resolution of symptoms
- gastroenteritis in children
    - red flags
        - appears to be unwell or deteriorating
        - altered responsiveness (e.g. irritable, lethargic)
        - sunken eyes
        - tachycardia
        - tachypnoea
        - reduced skin turgor
        - evidence of shock:
            - decreased level of consciousness
            - pale or mottled skin
            - cold extremities
            - tachycardia
            - tachypnoea
            - weak peripheral pulses
            - prolonged cap refill time
            - hypotension
    - differential
        - other sites of infection (e.g. UTI, otitis media, meningitis, pneumonia)
        - toddler's diarrhoea
        - constipation with overflow
        - acute appendicitis, mesenteric adenitis, malrotation of the gut
        - intussusception
        - coeliac disease
        - pyloric stenosis
        - regurgitation, posseting or GORD in babies
        - DKA
        - Addison's disease
    - fluid management
        - continue breastfeeding and other milk feeds

        - encourage fluid intake
        - discourage carbonated drinks
        - avoid fruit juices in severe dehydration, although dilute apple juice may be helpful
        - ORS for those at increased risk of dehydration
        - for children with clinical dehydration:
            - low-osmolarity ORS
            - 50ml/kg for fluid replacement over 4 hours as well as maintenance fluid given frequently and in small amounts
            - ORS may need to be given via NG tube if unable to drink or vomiting constantly
        - IV rehydration
            - 0.9% sodium chloride or 0.9% sodium chloride with 5% dextrose
            - 20ml/kg over 10 minutes for resuscitation
            - maintenance
                - 100ml/kg/day for first 10kg
                - 50ml/kg/day for next 10kg
                - 20ml/kg/day for the weight above 20kg
    - complications
        - increased risk of dehydration:
            - children <1 year, particularly <6 months
            - infants who were of low birth weight
            - >5 diarrhoeal stools in past 24h
            - >2 vomits in past 24h
            - not offered or unable to tolerate supplementary fluids
            - infants who have stopped breastfeeding during the illness
            - loss of lactase from the gut causing lactose intolerance

GC6 hepatitis

- differential diagnosis of hepatitis
    - acute
        - drugs, toxins, alcohol
        - infectious mononucleosis, CMV, Q fever, leptospirosis, syphilis, malaria, yellow fever
        - any other cause of jaundice
    - chronic
        - alcohol drugs
        - autoimmune hepatitis
        - haemochromatosis
        - Wilson's disease
- hepatitis A
    - background
        - most common form of acute viral hepatitis worldwide
        - RNA virus
        - usually spread by faecal-oral route, occasionally through food sources
        - direct correlation between increasing age and morbidity/mortality
        - incubation period is 2-6 weeks

        - after hepatocyte uptake, viral RNA is uncoated and host ribosomes bind to form polysomes; a viral RNA polymerase copies the viral genome; assembled viral particles are shed through the biliary tree into the faeces
            - shedding is greatest at 14-21 days after infection
    - risk factors
        - most people acquiring the virus do not have risk factors
        - personal contact
        - certain occupations (e.g. staff of large residential institutions, sewage workers)
        - travel to high risk areas
        - male homosexuality with multiple partners
        - IV drug abuse
        - people with clotting factor disorders receiving factor VIII and factor IX concentrates
    - clinical features
        - incubation period 2-6 weeks (mean 4)
        - prodrome of mild flu-like symptoms
            - anorexia, nausea, malaise, joint pain
        - smokers often lose their taste for tobacco
        - diarrhoea can occur, particularly in children
        - fever is not common
        - can progress to icteric phase:
            - dark urine (appears first)
            - pale stools (not always)
            - jaundice (70-85%)
            - abdominal pain (40%)
            - itch (usually with jaundice, can occur without)
            - arthralgias and skin rash
        - tender hepatomegaly, splenomegaly, lymphadenopathy
        - young children are usually asymptomatic
        - complete clinical recovery may take up to 6 months
    - investigations
        - IgM antibody to HAV
            - positive with onset of symptoms and for 3-6 (up to 12) months
                - sensitive and specific
        - IgG antibody to HAV
            - appears soon after IgM and persists for many years
            - in the absence of IgM represents past infection or vaccination
        - liver enzymes
            - ALT>AST
                - usually return to reference range over several weeks
            - ALP also rises
            - bilirubin rises
            - falls in serum albumin may occur
        - there may be low grade haemolysis and mild lymphocytosis
        - imaging
            - rarely, US to exclude other diseases

- management
  - mainly supportive
    - fluids
    - antiemetics
    - rest
  - avoid alcohol until liver enzymes normal
  - pregnant women should be advised or increased risk of miscarriage and premature labour
  - avoidance of food handling and unprotected sexual intercourse until non-infectious
  - outbreaks should be managed with advice from regional public health organisations
- complications (rare)
  - cholestatic hepatitis
    - severe pruritus, diarrhoea, weight loss, malabsorption
  - fulminant liver failure
  - relapsing HAV – can occur more than once
  - other very rare complications
    - AKI, red cell aplasia, Guillain-Barré syndrome, pancreatitis
- prognosis
  - excellent, usually self-limiting with no long-term sequelae
  - no carrier state and chronic liver disease does not occur

- hepatitis B
  - background
    - double-stranded DNA virus
    - most common cause of hepatitis worldwide
    - around 1 person in 350 is thought to have chronic hepatitis B infection in the UK
  - presentation
    - many new infections are subclinical or as a flu-like illness
    - incubation period ranges from 40-160 days
    - jaundice only occurs in 10% of younger children and 30-50% of adults
    - acute infection may rarely lead to fulminant hepatic necrosis, which is often fatal
    - illness usually starts insidiously with anorexia and nausea and an ache in the right upper quadrant
    - fever, when present, is usually mild
    - malaise may be profound, with disinclination to smoke or drink alcohol
    - as jaundice develops, there is progressive darkening of the urine and lightening of the faeces
    - presentation of decompensated liver disease includes ascites, encephalopathy and gastrointestinal haemorrhage
  - chronic hepatitis B
    - usually characterised by presence of detectable hepatitis B surface antigen (HBsAg) in the blood or serum for longer than 6 months
    - may be inactive and cause no significant health problems but may progress to liver fibrosis, cirrhosis and hepatocellular carcinoma

        - presence of HBeAg is associated with higher rates of viral replication and therefore increased infectivity
        - symptoms, if present, include:
            - fatigue, anorexia, nausea, right upper quadrant pain
        - risk of developing chronic hepatitis B infection is lowest in adults and increased in those with immunocompromise
        - chronic hepatitis B infection usually has a benign course in healthy non-drinkers with normal liver function
    - route of transmission
        - parenteral via blood or body fluids
            - vaginal or anal intercourse
            - sharing needles or needlestick injuries
            - blood transfusion (now rare in the UK as donations are screened)
            - vertical transmission
            - can be transmitted via human bites (rare)
    - investigations (usually under specialist care)
        - hepatitis B investigations
            - HBsAg, HBeAg, anti-HBe, anti-HBs, anti-HBcore
            - quantitative hepatitis B virus DNA
            - HBV genotype if considered for interferon
            - hepatitis delta virus serology
        - general liver investigations
            - FBC
            - bilirubin
            - liver enzymes
            - clotting
            - ferritin
            - lipid profile
            - autoantibody screen
            - caeruloplasmin
        - tests for hepatitis C and HIV
        - screening for liver cancer
        - ultrasonography
        - alpha-foetoprotein
    - management
        - general
            - avoid unprotected sexual intercourse until non-infectious or partners have received a full course of vaccination
                - contact tracing required
            - advise not to donate blood
            - advise on long-term effects of illness
        - treatment of acute infection
            - Health Protection notification
            - supportive treatment
                - fluids
                - antiemetics
                - rest

- avoid alcohol until liver enzymes normalise
- review current medications and stop non-essential medications
- itching can be difficult to treat
    - simple measures: stay cool, loose clothing, avoid hot baths and showers
    - chlorphenamine may help but should be avoided in severe liver impairment
- treatment with antivirals only indicated in fulminant hepatitis

▪ those with chronic disease should be referred to a hepatologist or gastroenterologist

- peginterferon alfa 2a is a treatment option for those with HbeAg-positive or -negative chronic hepatitis B and compensated liver disease
- surveillance for HCC with hepatic ultrasound and alfa-foetoprotein testing 6-monthly if significant fibrosis or cirrhosis or if older than 40 with family history of HCC and high HBV DNA

o complications

▪ fulminant hepatic failure
▪ relapse
▪ prolonged cholestasis
▪ chronic hepatitis
▪ cirrhosis (may require transplantation if decompensated)
▪ hepatocellular carcinoma
▪ glomerulonephritis
▪ cryoglobulinaemia

- hepatitis C
    - background
        - blood borne RNA virus
        - incubation period 6-9 weeks
        - infection may be acute or chronic and is usually asymptomatic when acute
        - routes of transmission include:
            - IV drug use
            - blood transfusion received before September 1991
            - haemodialysis
            - sexual contact with an infected individual
            - needlestick injuries in the healthcare setting
                - risk of transmission estimated at 3%
                - there is no post-exposure immunisation, antiviral treatment or immunoglobulin
                - repeated blood tests required if source known or suspected to be positive
            - perinatal transmission from an infected mother
                - breastfeeding is safe
            - other
                - poorly sterilised dental and medical equipment
                - tattooing, ear piercing, body piercing, acupuncture with unsterile equipment

        - sharing razors or toothbrushes contaminated with blood
    - chronic infection
        - about 75% develop chronic disease, with the remaining 25% clearing it at the acute stage
        - cirrhosis develops in 20-30% after 20 years
            - 1-4% of these patients develop hepatocellular carcinoma and 2-5% per year develop liver failure
    - factors associated with more rapid progression to severe liver disease
        - being over 40 at the time of infection
        - alcohol consumption
        - male gender
        - co-infection with HIV or hepatitis B
        - immunosuppressive therapy
    - risk factors for more aggressive disease
        - obesity
        - alcohol
        - diabetes
        - male
        - older age of acquiring infection
        - coinfection with HIV/hepatitis B
    - presentation
        - acute HCV infection
            - usually asymptomatic
            - 20-30% present with jaundice or deranged liver enzymes
            - 20-30% have non-specific symptoms such as anorexia, lethargy, abdominal pain
        - chronic HCV infection
            - indicated by fluctuating or elevated liver enzyme levels
            - often goes unrecognised for 10-20 years unless found incidentally
    - investigations include
        - HCV antibodies
            - if positive, HCV RNA to check for active infection
            - repeated antibody testing at intervals if negative antibody testing
        - other baseline investigations
            - FBC, U&Es, LFTs, clotting, HbA1c, TFTs, ferritin
            - hepatitis A and B serology and HIV testing
            - screening for other STIs if indicated
    - associated diseases
        - increased risk of:
            - diabetes mellitus
            - Sjögren's syndrome
            - essential mixed cryoglobulinaemia
            - polyarteritis nodosa
            - autoimmune hepatitis
            - thyroiditis
            - membranous glomerulonephritis
            - porphyria cutanea tarda

                - lichen planus
                - immune thrombocytopenia
        - management
            - notification of local Health Protection team
            - for acute infection, if needed, interferon can be started at 3-6 months after diagnosis – increases chance of clearing disease
            - chronic HCV requires specialist management and monitoring for HCC
                - antiviral combination therapy usually consists of weekly self-administered subcutaneous pegylated interferon alfa and daily doses of oral ribavirin
                - liver transplantation may be required in end-stage liver disease; recurrence of HCV is common post-transplant
            - lifestyle advice
                - stop alcohol and smoking
                - healthy body weight and diet
                - counselling
                - advice not to donate blood or organs, tissues or semen or share razors, toothbrushes or other objects that may be contaminated with blood
                - risk of sexual transmission is reduced in a stable relationship but greater with risky sexual practices
                - the patient should be encouraged to inform injecting or sexual contacts so they can be tested
            - patients should be offered hepatitis A and B immunisation
        - complications
            - hepatocellular carcinoma in 1-5%
                - suggested by weight loss and raised alpha-fetoprotein
        - prognosis
            - rate of progression is slow but variable
            - HCV infection is cured in more than 99% of people who achieve a sustained viral response
- hepatitis D
    - background
        - unusual, defective, single-stranded RNA virus
        - requires the presence of HBV to replicate
        - can be acquired along with or after HBV
        - transmission is by infected blood and blood products, percutaneously and sexually
            - perinatal transmission is rare
    - clinical features
        - may increase risk of severe clinical hepatitis, fulminant hepatic failure, chronic liver disease, cirrhosis and hepatocellular carcinoma in HBV patients
    - investigations
        - anti-HDV antibody plus other investigations as indicated for hepatitis, liver failure, cirrhosis and HCC
    - management
        - includes pegylated interferon alfa and liver transplantation (can be curative)

        - management is otherwise supportive
- hepatitis E
    - background
        - some genotypes can cause liver disease in humans
        - infection may very in severity from inapparent to fulminant
        - mortality is 1-4% (up to 25% in pregnant women)
        - spreads mainly though contamination of water supplies in resource-limited coutries
        - transmission from domestic pigs to humans is common
    - route of transmission
        - faecal-oral route (similar to HAV)
        - contaminated drinking water is the most common source
        - person-to person transmission is rare; maternal-neonatal transmission does occur
        - zoonotic spread may occur
    - clinical features
        - similar to HAV with no apparent risk of chronic liver disease
        - incubation 2-9 weeks
        - usually self-limiting
        - fulminant disease occurs in around 10%
    - investigations
        - hepatitis serology
    - management
        - mainly supportive as for HAV
    - prevention
        - good hygiene and sanitation
        - avoidance of tap water in high risk areas
- drug-induced hepatitis
    - background
        - inflammation of the liver caused by medication
        - parenchymal destruction tends to be more extensive than in acute viral hepatitis
        - 25-50% of cases of hepatitis may be due to adverse drug effects
        - risk factors include genetic predisposition, age, gender, pre-existing liver disease and comorbidities
        - hepatotoxicity can be predictable or unpredictable
        - drugs affect the liver in different ways:
            - acute hepatocellular damage
                - dose-unrelated
                    - e.g. antituberculous drugs, halothane, anticonvulsants
                - dose-related
                    - e.g. alcohol, paracetamol poisoning, amiodarone, methotrexate
                - both dose-unrelated and dose-related
                    - e.g. azathioprine
            - chronic active hepatitis
                - e.g. isoniazid, nitrofurantoin

- cirrhosis
    - e.g. alcohol, methotrexate
- hepatic tumours
    - e.g. anabolic steroids, combined oral contraceptives
- intrahepatic cholestasis
    - either dose unrelated (e.g. carbimazole, erythromycin, phenothiazines) or dose-related (e.g. anabolic steroids, azathioprine, oestrogens)
- gallstones
    - e.g. clofibrate, oestrogens

- presentation
    - may be asymptomatic and detected by routine monitoring
    - symptoms and signs are similar to other causes of liver damage
    - onset is usually abrupt with chills, fever, rash, pruritus, arthralgia, headache, abdominal pain, anorexia, nausea and vomiting
    - later, overt evidence of liver damage may develop, e.g. jaundice, dark urine, enlarged and tender liver
    - two general pathogenic mechanisms:
        - predictable or direct
            - usually promptly follows exposure to a new medication
            - mechanism appears to be due to direct toxicity or a toxic metabolite
        - unpredictable or idiosyncratic
            - may be related to immune hypersensitivity
            - rash, fever and eosinophilia are typically present
            - reactions follow exposure by a few weeks (e.g. co-amoxiclav)
            - late onset idiosyncratic reactions are difficult to recognise
                - follow exposure by a few months and usually do not display features of hypersensitivity
- management
    - no specific treatment other than discontinuing the medication causing the problem
    - people with acute hepatitis should avoid physical exertion, alcohol, paracetamol and any other hepatotoxic substances
    - there are no specific antidotes other than NAC for paracetamol toxicity
    - supportive care for acute liver failure and consideration of transplantation if required

- autoimmune hepatitis
    - background
        - rare and chronic liver disease
        - characterised by increased serum transaminases and immunoglobulin G, inflammatory liver histology and presence of circulating autoantibodies
    - presentation
        - can be acute, fulminant, asymptomatic or chronic
        - subclinical disease often precedes the onset of symptoms
        - common symptoms include:

                - fatigue, myalgia, mild pruritus
                - nausea (often a prominent symptom)
                - upper abdominal discomfort
                - anorexia, diarrhoea
                - arthralgias
                - skin rashes (including acne), hirsutism
                - oedema
                - amenorrhoea
                - chest pain (pleuritis)
                - weight loss and intense pruritus (unusual)
            - signs
                - hepatomegaly
                - jaundice (around 50%)
                - splenomegaly
                - spider angiomata
                - ascites
                - encephalopathy
    - investigations
        - exclusion of common viral, drug-induced and metabolic liver disease
        - autoantibodies
        - serum protein electrophoresis and quantitative immunoglobulins
        - aminotransferases
        - FBC and blood film
        - liver biopsy
            - confirms disease and provides information on prognosis
    - management
        - lifelong treatment to prevent development of cirrhosis and end-stage liver disease
        - steroid induction followed by maintenance therapy with azathioprine – effective in most cases
            - other immunosuppressants can be tried if needed
    - monitoring
        - testing for hepatitis A and B immunity and vaccination if needed
        - regular blood testing including LFTs, glucose and FBC
        - calcium and vitamin D supplements
        - DXA scan prior to starting steroids and at 1-2 yearly intervals
        - screening for glaucoma and cataracts after 12 months of prednisolone treatment
    - complications
        - hyperviscosity syndrome secondary to high IgG levels
        - hepatocellular carcinoma, more common in patients with cirrhosis
    - prognosis
        - without treatment, nearly 50% die in approximately 5 years
        - outlook for treated patients is generally good
        - cirrhosis develops in up to 50%

GC7 inflammatory bowel disease

- Crohn's disease
    - background
        - chronic relapsing inflammatory bowel disease
        - characterised by a transmural granulomatous inflammation which can affect any part of the gastrointestinal tract (most commonly ileum, colon or both)
        - there may be skip lesions (unaffected bowel between areas of active disease
        - extra-intestinal manifestations include iritis, arthritis, erythema nodosum and pyoderma gangrenosum
        - prevalence in the UK is around 145 per 100,000 population
        - equal sex distribution
    - risk factors
        - genetics/family history
        - smoking (increases risk 3-4 fold; smokers tend to have more aggressive disease and earlier post-operative relapse)
        - other exacerbating factors include intercurrent infection and NSAIDs
    - presentation
        - symptoms
            - variable, often include diarrhoea (may be bloody and become chronic), abdominal pain and/or weight loss
            - typically there are periods of acute exacerbation interspersed with remissions
            - systemic symptoms of malaise, anorexia of fever are common
            - children may present with poor growth, delayed puberty, malnutrition and bone demineralisation
    - examination
        - general ill health with signs of weight loss, fluid depletion and anaemia
        - during acute exacerbations – hypotension, tachycardia and pyrexia
        - abdominal tenderness or distension, palpable masses
        - anal and perianal lesions (pendulous skin tags, abscesses, fistulae)
        - mouth ulcers
        - other extra-intestinal features
            - clubbing, erythema nodosum, pyoderma gangrenosum
            - conjunctivitis, episcleritis, iritis
            - large joint arthritis, sacroiliitis, ankylosing spondylitis
            - fatty liver, primary sclerosing cholangitis (rare), cholangiocarcinoma (rare)
            - granulomata may occur in the skin, epiglottis, mouth, vocal cords, liver, nodes, mesentery, peritoneum, bones, joints, muscle or kidney
        - renal stones
        - osteomalacia
        - malnutrition
        - amyloidosis
    - investigations
        - clinical evaluation plus combination of endoscopic, histological, radiological and biochemical investigations
        - bloods include FBC, U&Es, LFTs and CRP

        - faecal calprotectin
            - recommended when considering a differential of IBD and irritable bowel syndrome
            - a normal level has a very high negative protective value with a positive value then potentially requiring colonoscopy
        - microbiological testing for infectious diarrhoea
        - endoscopy and biopsy
    - differential
        - infectious gastroenteritis
        - tuberculosis
        - ulcerative colitis
        - actinomycosis
        - carcinoid
        - amyloidosis
        - intestinal lymphoma
        - Behçet's disease
        - bowel carcinoma
        - ischaemic colitis
        - radiation or drug-induced colitis
        - diverticulitis
        - coeliac disease
        - irritable bowel syndrome
        - acute ileitis may mimic acute appendicitis
    - management
        - rule out of cancer, particularly in patients over 50
        - steroids are usually helpful for extra-articular manifestations
        - consider admission if:
            - severe abdominal pain, especially if there is tenderness on palpation
            - severe diarrhoea (8+ times/day) with or without rectal bleeding
            - bowel obstruction
            - fever and systemically unwell
        - NICE guidance
            - inducing remission
                - monotherapy with conventional glucocorticosteroid (prednisolone, methylprednisolone or IV hydrocortisone) for first presentation or single exacerbation in 12 months
                - budesonide and 5-ASA are alternatives
            - add-on treatments
                - azathioprine, mercaptopurine, methotrexate, infliximab, adalimumab
            - maintaining remission
                - smoking cessation
                - azathioprine or mercaptopurine or methotrexate
                - post-operative metronidazole
            - surgery
                - may be offered early if disease limited to distal ileum
                - balloon dilatation for management of strictures

        - fistulas may require exploration and drainage
    - complications
        - bowel
            - strictures causing subacute or acute obstruction
            - fistula between bowel loops, bladder, vagina, skin
            - perforation, acute dilatation, massive haemorrhage
            - increased risk of colonic carcinoma
        - osteoporosis
        - renal disease
            - secondary to obstruction of right ureter by ileocaecal disease
        - iron, folate and B12 deficiency
        - gallstones and renal stones
    - prognosis
        - more than 50% need surgery within 10 years of diagnosis
        - 30% have a fairly indolent disease course
        - clinical indicators of poor prognosis at diagnosis include perianal or stricturing disease, weight loss >5kg or the need for steroids
- ulcerative colitis
    - background
        - idiopathic chronic inflammatory disease of the colon that follows a course of relapse and remission
        - most common cause of inflammatory bowel disease
        - aetiology is unknown
        - risk is decreased in smokers
    - symptoms
        - cardinal symptom is bloody diarrhoea
        - associated symptoms include: colicky abdominal pain, urgency, tenesmus
        - proctitis may present with constipation and rectal bleeding
        - systemic symptoms may be present including malaise, fever, weight loss and extra-intestinal symptoms (joint, cutaneous, eye)
    - signs
        - depend on disease severity
        - patient may be unwell, pale, febrile, dehydrated, tachycardic, hypotensive
        - abdominal examination may reveal tenderness, distension, palpable mass
    - extra-intestinal disease (around 4% of patients)
        - related to activity of the colitis
            - erythema nodosum
            - aphthous ulcers
            - episcleritis
            - acute arthropathy
        - usually related to activity of colitis
            - pyoderma gangrenosum
            - anterior uveitis
        - not related to activity of colitis
            - sacroiliitis
            - ankylosing spondylitis
            - primary sclerosing cholangitis

- differential
  - Crohn's disease
  - prolonged use of laxatives
  - infective colitis
    - chronic schistosomiasis, amoebiasis, tuberculosis
  - mild colitis may mimic irritable bowel syndrome
  - other conditions:
    - ischaemic colitis
    - radiation colitis
    - bowel trauma
    - colorectal cancer
    - diverticulitis
    - polyposis syndromes
    - colonic polyps
- investigations
  - clinical suspicion plus macroscopic findings on sigmoidoscopy or colonoscopy, typical histological findings on biopsy and negative stool examinations for infective agents
  - bloods including FBC, U&Es, LFTs, CRP, iron studies, vitamin B12, folate
  - faecal calprotectin
  - imaging may include abdominal US, CT, MRI, barium fluoroscopy and isotope-labelled scans
- disease severity (NICE)
  - mild
    - fewer than four stools/day, no more than small amounts of blood in stools, no anaemia, HR <90, no fever, normal ESR/CRP
  - moderate
    - four to six stools/day with more blood than mild disease; no anaemia, HR <90, no fever, normal ESR/CRP
  - severe
    - six or more stools/day, visible blood in stools and at least one feature of systemic upset (temperature above 37.8 degrees, HR >90, anaemia, ESR >30)
- consideration for admission
  - patients with severe colitis
  - patients with moderate disease who fail to respond to steroids within two weeks
- management
  - topical management may be adequate for some patients, particularly with proctitis
  - oral or parenteral therapy is used for patients with more extensive disease
  - leukophoresis (extracorporeal removal of leucocytes from the blood) for selected patients
  - antimotility and antispasmodic drugs should not be used and may precipitate paralytic ileus and megacolon in active ulcerative colitis
  - drug treatments
    - aminosalicylates

        - mesalazine (5-ASA) is the treatment of choice for induction and maintenance or mild-to-moderate ulcerative colitis
    - corticosteroids
        - used to induce remission in relapses with no role in maintenance therapy
        - may be topical (suppositories, liquid or foam enemas), oral or IV
    - thiopurines
        - azathioprine and its metabolite 6-mercaptopurine may be used when:
            - patients intolerant to steroids
            - two or more corticosteroid courses required in a year
            - relapses when prednisolone dose is less than 15mg/day
            - relapses within 6 weeks of stopping steroids
    - others
        - ciclosporin, infliximab, adalimumab, golimumab, vedolizumab, stool bulking agents
    - surgery
        - 20-30% of patients ultimately require surgery
            - colectomy is curative
            - usual procedure is a restorative proctocolectomy with ileal pouch-anal anastomosis
        - there may be an increased likelihood of needing surgery for patients with:
            - stool frequency of >8/day
            - pyrexia
            - tachycardia
            - abdominal x-ray showing colonic dilatation
            - low albumin, low haemoglobin, high platelet count or CRP >45
- complications
    - colorectal cancer
        - about double the incidence
        - colonoscopic surveillance is recommended
    - pouchitis
        - generally managed with metronidazole or ciprofloxacin
    - post-IPAA complications
        - leakage
        - pelvic abscess
    - toxic megacolon
        - may be triggered by hypokalaemia, opiates, anticholinergics, barium enemas
        - initial management is IV fluids, IV steroids, antibiotics and IV ciclosporin
        - total colectomy may be required

        - osteoporosis
        - psychosocial and sexual problems
    - prognosis
        - lifelong condition, with unpredictable relapses and remissions
        - factors which may suggest a poor prognosis include:
            - severe symptoms at presentation
            - extensive disease
            - raised inflammatory markers
            - age <50, especially childhood-onset disease
            - poor compliance with drug treatment

GC8 peptic ulcer disease

- background
    - 10% of the population of industrialised countries are likely to develop a duodenal or gastric ulcer in their lifetime
    - peptic ulcer disease is the most common cause of upper GI bleeding worldwide
    - definition
        - ulcers are defined as mucosal erosions greater than or equal to 0.5cm
        - peptic ulcers occur in the gastrointestinal tract and are characterised by mucosal damage from pepsin and gastric acid secretion
        - common sites:
            - stomach
            - proximal duodenum
        - less common sites:
            - lower oesophagus (e.g. within a sliding hiatus hernia)
            - distal duodenum
            - ileum (e.g. Meckel's diverticulum)
- pathophysiology
    - causes:
        - Helicobacter pylori
            - Gram-negative, motile, spiral-shaped bacterium
            - over half of the population of the world is thought to be infected
                - less than 20% ever demonstrate clinical gastrointestinal disease
            - H. pylorus lives in the mucous layer above gastric surface epithelial cells
            - multiplies optimally at a pH of 6.0-8.0
                - it converts urea produced from gastric cells into ammonia and bicarbonate to buffer gastric acid
                - the ammonia prevents D cells (which would normally release somatostatin in response to low antral pH) from sensing the true pH, allowing excess acid production
            - H. pylorus can only colonise gastric epithelial cells, however excess acid production can provoke gastric metaplasia of duodenal cells, allowing colonisation
            - the immune response provoked by H pylori also impairs mucosal defences and can lead to ulceration

        - NSAIDs
            - up to 20% of peptic ulcer disease is attributable to NSAIDs
            - ulcers and ulcer-related complications increase with older age, concurrent steroid or anticoagulant use, prior ulcer or bleeding and major organ impairment
            - mucosal injury is caused by multiple mechanisms
                - inhibition of prostaglandin synthesis by inhibition of cyclooxygenase-1
                    - can lead to reduced mucous and bicarbonate secretion, reduced mucosal blood flow and impaired platelet aggregation, reducing host defences to injury
            - NSAIDs also exert a direct local toxic effect to gastric cells
        - cigarette smoking
            - smokers are twice as likely to develop peptic ulcer disease
            - increases gastric acid secretion, duodenogastric reflux and decreases prostaglandin and bicarbonate production
        - medications
            - alcohol
            - steroids
            - potassium chloride
            - bisphosphonates
            - chemotherapy agents
        - Zollinger-Ellison syndrome
            - a gastrin-secreting tumour can cause increased gastric acid secretion in addition to parietal cell hyperplasia, resulting in peptic ulcer disease
        - Meckel's diverticulum
            - caused by incomplete obliteration of the vitelline duct
            - ectopic gastric mucosa present in the distal ileum can lead to ulceration and complications such as bleeding and perforation
        - others:
            - neoplastic lesions (primary or secondary)
            - Crohn's disease
            - viral infections, especially if immunocompromised (CMV, HSV)
            - post-partial gastrectomy
            - radiation damage
            - stress
                - physiological (e.g. burns, major trauma)
                - psychological
- history
    - the most specific and classic features are:
        - a history of episodic epigastric pain
        - relief of pain after food intake
        - night-time awakening because of relief with food intake
    - epigastric pain is often described as gnawing or burning
    - duodenal ulcers are typically worsened by hunger/fasting and relieved by eating

        - gastric ulcers may conversely cause pain on eating and fear of eating due to pain may cause weight loss
    - less commonly, there may be nausea, vomiting, abdominal bloating/fullness, early satiety, heartburn or intolerance of fatty foods
    - patients may present initially with complications such as bleeding or perforation
        - absence of other symptoms is more likely with NSAID-induced ulcers
    - patients should be asked about melaena/rectal bleeding, coffee-ground vomiting or haematemesis
- examination
    - physical finding are uncommon
    - alarm features:
        - dysphagia
        - evidence of GI blood loss
        - unexplained weight loss
        - upper abdominal mass
        - persistent vomiting
- investigations
    - baseline observations including postural BP
    - urinalysis and hCG in women of childbearing age
    - FBC, U&Es
        - low Hb and raised urea may suggest bleeding
    - LFTs, amylase
    - G&S or crossmatch if suspicion of bleeding
    - 12 lead ECG to look for evidence of ACS as cause
    - CXR
- management
    - Emergency Department goals
        - identify patients with life-threatening complications and/or haemodynamic instability and initiate resuscitation
        - identify patients with suspected peptic ulcer disease who require admission or urgent referral
        - identify patients with suspected peptic ulcer disease who can be safely discharged from the ED
        - arrange appropriate follow up for those who are discharged
    - bleeding
        - it may be appropriate to consider high dose PPI pre-endoscopy in high-risk patients (e.g. pantoprazole 40mg IV), particularly if there is likely to be a delay to endoscopy
        - bleeding may be subtle and should be actively sought in history, examination and investigations
    - perforation
        - occurs in 2-10% of patients with peptic ulcer disease
        - normally involves the anterior wall of the duodenum (60%)
            - may also affect the gastric antrum (20%) and lesser curvature (20%)
        - resultant bacterial peritonitis has a mortality of 30-50% in older patients
        - clinical features:
            - often sudden, severe abdominal pain which rapidly worsens

- pain may radiate to the back or either shoulder
- generalised abdominal tenderness with guarding, rebound tenderness and/or rigidity with hypoactive bowel sounds may be present
- patients may be tachycardic and hypotensive with fever and/or oliguria

- erect CXR may show subdiaphragmatic air but may be normal and CT or surgery may be required depending on clinical state and likelihood of perforation
- management is resuscitation plus broad spectrum IV antibiotics
- surgical intervention is normally laparotomy, placement of an omental patch and washout of peritoneal contents
- perorated gastric ulcers may be treated with omental patch, wedge resection of the ulcer or partial gastrectomy and reanastamosis

- gastric outlet obstruction
  - peptic ulcer disease accounts for 5-8% of cases with pyloric stenosis common in childhood and neoplastic lesions in the older population
  - clinical features:
    - recurrent large-volume vomiting containing undigested food
    - dehydration
    - abdominal distension
      - often tympanic and may be associated with a palpable epigastric mass +/- visible peristalsis
    - weight loss, lethargy, malaise
    - there may be a hypochloraemic, hypokalaemic metabolic alkalosis
  - management
    - resuscitation
    - CXR and AXR
    - NG tube
    - surgical review
      - endoscopy to assess site, severity and nature of disease
      - surgical removal of the obstructive lesion may be possible
      - malignancy should be actively investigated
    - obstruction caused by inflammation and oedema due to peptic ulcer disease usually responds to H pylori eradication and PPIs
- patients with no haemodynamic instability or complications
  - suspicion of neoplasm can usually be managed with urgent outpatient investigation
  - for patients with uncomplicated dyspepsia and no alarm features an initial approach of lifestyle advice (decrease or stop smoking/alcohol/illicit drug use; weight reduction; healthy eating advice) is recommended
  - if symptoms fail to resolve, options are:
    - trial of PPI or H2 receptor antagonist for 1 month
    - test and treat
      - non-invasive H pylori test, eradication of infection in those who test positive and symptomatic relief for those who test negative

        - patients must be at least 2 weeks free of PPI and 4 weeks free of antibiotic therapy before testing to avoid false negatives
        - H pylori non-invasive tests are:
            - 13C-urea breath test (CUBT) and faecal antigen test
            - tests confirm active infection and are also useful for retesting post-eradication
        - in patients who have endoscopy, biopsy specimens can be tested for H pylori
        - if test results are positive, patients should be treated with antibiotics and acid-inhibitors (usually a PPI and two antibiotics)
            - if successful and patient remains asymptomatic with no alarm features, endoscopy is not required
        - if test results are negative, the possibility of NSAID-associated ulcer should be considered
            - if NSAIDs cannot be discontinued, PPI or H2 receptor antagonist trial for 1 month
            - prostaglandin analogues such as misoprostol may be considered
    - endoscopy
        - aims:
            - haemorrhage control in patients with upper GI bleeding
            - confirmation of peptic ulcer disease in patients with persistent dyspepsia
            - biopsy for confirmation of H pylori infection and/or neoplasia
            - reassessment in patients with ongoing symptoms despite H pylori eradication or withdrawal of NSAIDs

GC9 pyloric stenosis

- background
    - caused by diffuse hypertrophy and hyperplasia of the smooth muscle of the antrum of the stomach and pylorus, resulting in narrowing of the pyloric canal, which can cause obstruction
    - there is a familial link
    - more common in white populations
- clinical features:
    - commoner in first born males
    - 80% males
    - 10% are premature
    - projectile, non-bilious vomiting in neonate
        - usually 30-60 minutes after a feed
        - baby remains hungry
    - 2-8 weeks
    - dehydration
    - weight loss

    - visible peristalsis and olive-sized mass in epigastrium (best palpated at start of feed on or to right of midline when liver is displaced superiorly)
    - confirmed with US
    - associated pathology – cleft palate, GORD
- biochemistry
    - hypochloraemia
        - loss of chloride in vomitus
    - metabolic alkalosis
        - loss of H+ in vomitus
        - decreased secretion of pancreatic bicarbonate
        - increased bicarbonate presented to distal tubule and eliminated, producing an alkaline urine
    - hyponatraemia
        - loss of sodium in vomitus
        - decreased absorption of sodium
        - loss of sodium in urine until kidney adjusts to increased bicarbonate load
        - activation of renin-AG-ALD system to offset this and restore sodium and water
    - hypokalaemia
        - loss of potassium in vomitus
        - activation of renin-AG-ALD system with loss of potassium in urine
        - with extreme potassium loss in urine, it gets reabsorbed in the distal tubule with loss of H+, worsening metabolic alkalosis and producing acidic urine
    - initially, alkaline urine, later paradoxical acidic urine
        - to prevent hypokalaemia
    - dehydration
        - inability to absorb enteral fluid and vomiting
        - activation of renin-AG-ALD system and ADH
- management
    - fluid resuscitation
        - determined by weight and degree of clinically assessed dehydration (tissue turgor, pulse, fontanelle, central capillary refill, peripheral perfusion, respiratory rate)
            - IV boluses of 0.9% sodium chloride (10-20ml/kg) to restore circulating volume
            - maintenance at 4ml/kg/hr with 5% dextrose and 0.45% sodium chloride and 20mmol KCl
            - titrate as needed (aim 2ml/kg/hr of urine)
            - need a lot of potassium once they pee
    - laboratory criteria by which patient is sufficiently resuscitated for surgery:
        - serum Cl^- of at least 105mmol/L
        - serum HCO_3^- normal
        - urinary Cl^- of >20mmol/L
    - intra-operative
        - myomectomy
            - splitting of the pylorus muscle longitudinally down to the mucosa
        - risk of pulmonary aspiration from gastric outflow obstruction

                - NG should be aspirated and left in situ to help decompress stomach from vigorous ventilation
            - RSI
            - extubation awake and in left lateral position
        - post-operative
            - NG tube removal
            - feeding within 6 hours
            - maintenance IV fluids until feeding established
            - apnoea alarm used overnight

HAEMATOLOGY

HP1 anaemia

- definition:
    - <120g/L in females, <130g/L in males (WHO definition)
- pathophysiology:
    - erythropoiesis
        - tissue hypoxia stimulates EPO release by kidneys
        - EPO stimulates bone marrow erythropoiesis if adequate supplies of iron, B12, folate
        - reticulocytes may be seen with rapid erythropoietic response
    - RBC lifespan
        - 120 days, reduced in disease states
        - removed by reticuloendothelial system (including spleen and liver)
    - effects of anaemia
        - impaired oxygen delivery
        - decreased blood viscosity
    - response
        - increased cardiac output
            - reduced resistance to blood flow (reduced viscosity)
            - increased flow to and from heart (increased preload, decreased afterload)
            - minor role for increased heart rate/myocardial contractility if normovolaemia maintained
        - oxygen extraction ratio increases
            - redistribution of blood flow to areas of high demand (e.g. myocardium, brain)
            - capillary blood flow increased, pre-capillary oxygen loss reduced
        - 2,3-DPG increases
            - occurs in chronic anaemia
            - promotes offloading of oxygen to tissues (right shift of dissociation curve)
- mechanisms:
    - reduced RBC production
        - nutritional deficiency, malabsorption (iron, folate, B12)

        - bone marrow failure (infiltration, myelodysplasia, chemotherapy, radiotherapy)
        - low levels of stimulating hormones (EPO, thyroid hormones)
    - increased RBC destruction
        - haemolytic anaemia
    - RBC loss
- investigations:
    - MCV
    - blood film
    - reticulocytes
    - B12 and folate
    - iron studies (ferritin, iron, total iron binding capacity)
        - normal ferritin does not exclude deficiency in critically ill as it rises in inflammation
    - haemolysis screen (haptoglobins, Coombs' test, urine Hb, LDH, autoimmune screen)
    - Hb electrophoresis
    - bone marrow biopsy
    - LFTs
    - TFTs
    - lead level if basophilic stippling on film
    - consider septic screen
    - coagulation profile
- management:
    - find and treat cause
    - find and control bleeding, treat coagulopathy
    - ensure adequate oxygen delivery
    - consider EPO, RBC, iron supplementation
    - iron, B12, folate supplements if indicated
    - consider haematology referral
    - transfusion:
        - evidence is limited for benefits, significant risks
        - restrictive strategy - transfuse if <70g/L
        - anaemia less tolerated in older patients, severely ill, coronary, respiratory or cerebrovascular disease – consider earlier transfusion
- MCV:
    - decreased
        - iron deficiency
        - thalassaemia
        - haemoglobinopathies
        - sideroblastic anaemia
    - normal
        - pregnancy
        - haemorrhage
        - haemolysis
        - renal failure
        - malignancy
        - anaemia of chronic disease
        - bone marrow failure

    - increased
        - B12/folate deficiency
        - alcohol
        - liver disease
        - thyroid disease
        - myelodysplasia
        - anti-folate drugs (e.g. methotrexate)
- blood film abnormalities:
    - acanthocytes
        - irregularly shaped RBCs
        - liver disease
    - anisocytosis
        - RBCs of various sizes
        - megaloblastic anaemia, thalassaemia
    - blast cells
        - nucleated precursor cells
        - myelofibrosis, leukaemia
    - echinocytes
        - spiculated RBCs seen in renal failure
    - Howell-Jolly bodies
        - nuclear remnants in RBCs
        - post splenectomy
    - hypochromic
        - pale RBCs
        - iron-deficiency anaemia
    - left shift
        - immature white cells
        - infection, marrow infiltration
    - leukaemoid reaction
        - reactive leucocytosis
        - infection, burns, haemolysis
    - poikilocytes
        - variably shaped cells
        - iron or B12 deficiency
    - reticulocytes
        - immature RBCs
        - haemorrhage, haemolysis
    - right shift
        - hypersegmented neutrophils
        - megaloblastic anaemia, liver disease
    - rouleaux
        - clumping of RBCs
        - infection, inflammation
    - schistocytes
        - fragmented RBCs
        - microangiopathic haemolytic anaemia
    - spherocytes
        - spherical RBCs

        - autoimmune or hereditary
    - target cells
        - RBCs with central staining and outer pallor
        - liver disease, haemoglobinopathy

HP2 bruising and spontaneous bleeding

- purpuric rashes
    - background
        - purplish discoloration of the skin produced by small bleeding vessels near the surface
        - may occur in the mucous membranes, especially of the mouth and internal organs
        - may present as petechiae, purpura or ecchymoses
        - may be non-thrombocytopenic (normal platelet count) or thrombocytopenic
    - history
        - age of patient
            - HSP tends to occur in children
            - senile purpura is confined to the elderly
        - duration of rash and whether it is changing noticeably
        - whether the patient is otherwise well
        - whether general easy bruising has been noticed
        - review of recent travel history
    - differential
        - non-thrombocytopenic purpura
            - congenital causes
                - hereditary haemorrhagic telangiectasia (Osler-Weber-Rendu syndrome)
                - connective tissue diseases (e.g. Ehlers Danlos)
                - congenital CMV or rubella
            - acquired causes
                - e.g. severe infections such as septicaemia, meningococcus, measles
            - allergic causes
                - e.g. HSP, connective tissue disorders, SLE
            - drug induced causes
                - e.g. steroids, sulfonamides
            - other causes
                - senile purpura
                - trauma
                - scurvy
                - dependent purpura with venous hypertension
                - factitial purpura
        - thrombocytopenic purpura
            - impaired platelet production
            - excessive platelet destruction
            - sequestration of platelets
            - dilutional loss

- investigations
  - FBC
  - ESR/CRP
  - LFTs
  - coagulation screen
  - consider plasma electrophoresis
  - autoantibody screen for connective tissue disorders
- diseases associated with purpuric rashes
  - bacterial infections
    - meningococcal and streptococcal septicaemia
    - diphtheria
    - smallpox
    - chickenpox
    - measles
    - parvovirus B19
    - haemorrhagic fevers
  - allergic vasculitic purpura
    - e.g. HSP
    - caused by inflammation and infiltration of the blood vessel wall
    - HSP is often preceded by an URTI caused by beta-haemolytic streptococcus
      - typically affects front of legs and buttocks
      - may be associated with acute arthritis, gastrointestinal pain and nephritis with proteinuria
      - the purpuric rash may continue to form over several weeks
      - serious acute complications include:
        - CNS bleeding
        - acute intussusception
        - AKI
      - usually self-limiting
  - disseminated intravascular coagulation
    - massive ecchymosis with sharp irregular borders and an erythematous halo
    - can evolve to haemorrhagic bullae and blue-black gangrene
    - appears as multiple lesions, often symmetrically, involving distal extremities, areas of pressure, lips, ears, nose and trunk
  - strong steroids
    - caused by atrophy of collagen fibres supporting blood vessels in the skin
  - blood transfusions
    - usually confined to multiparous women
  - pigmented purpuric dermatoses
  - amyloid
  - factitial purpura
- thrombocytopenia and platelet function disorders
  - background

        - thrombocytopenia is a reduction in the platelet count below the normal lower limit (usually 150 x 10^9/L)
        - risk of bleeding is not based on platelet count alone
    - causes
        - disorders affecting platelet production
            - congenital
                - megakaryocytic hypoplasia
                    - underdevelopment of megakaryocytes which normally develop in bone marrow and fragment to produce platelets
                    - usually autoimmune or infectious origin
                - thrombocytopenia/absent radii (TAR syndrome)
                    - radial aplasia or hypoplasia and thrombocytopenia
                - Bernard-Soulier syndrome
                - Wiskott-Aldrich syndrome
                    - X-linked recessive disease characterised by thrombocytopenia, lymphopenia and depressed cellular immunity, eczema, malignant lymphoma
                - May-Hegglin anomaly
                    - thrombocytopenia, giant platelets and leucocyte inclusions
                - congenital leukaemia
                    - e.g. in association with Down's syndrome
                - Fanconi's anaemia
            - decreased production (disorders of bone marrow)
                - viral infections
                    - e.g. herpes simplex, CMV, varicella-zoster, EBV, rubella, enterovirus, mumps, hepatitis, HIV
                - aplastic anaemia
                - marrow infiltration by a malignancy
                    - e.g. leukaemia, lymphoma, myeloma, metastatic malignant disease
                - drugs
                    - e.g. chemotherapy
                - alcohol
                - paroxysmal nocturnal haemoglobinuria
                - megaloblastic anaemia
                - myelofibrosis
                - miliary tuberculosis
        - decreased platelet survival
            - immune
                - immune thrombocytopenia
                - SLE
                - rheumatoid arthritis
                - sarcoidosis
                - antiphospholipid syndrome
            - post-transfusion thrombocytopenic purpura

        - antigens on transfused platelets can lead to destruction of not just the transfused platelets but the patient's own too
        - starts about 10 days after transfusion but can last several weeks or months
    - neonatal alloimmune thrombocytopenia
        - occurs when the mother produces antibodies against fetal platelets with paternal antigens
        - most common cause of severe neonatal thrombocytopenia
        - often follows an apparently uneventful pregnancy but has high risk of intracranial haemorrhage and high mortality
        - commonly occurs in first pregnancies, unlike haemolytic disease of the newborn
    - drug induced
        - heparin
        - carbamazepine
        - ibuprofen
        - quinidine
        - quinine
        - rifampin
        - sulfamethoxazole
        - trimethoprim
        - vancomycin
    - thrombotic thrombocytopenic purpura
    - haemolytic uraemic syndrome
    - disseminated intravascular coagulation
    - HELLP syndrome in pregnancy
        - **h**aemolysis
        - **e**levated **l**iver enzymes
        - **l**ow **p**latelet count
    - cardiopulmonary bypass
    - splenomegaly and hypersplenism
        - may be associated with a variety of conditions such as cirrhosis, malaria, lymphoma
    - Kasabach-Merritt syndrome
        - cavernous haemangiomata with severe thrombocytopenia and features of DIC
- dilutional thrombocytopenia
    - caused by transfusion of large volumes of blood which may be depleted of functioning platelets, resulting from prolonged storage
- platelet function disorders
    - inherited platelet function disorders
        - severe disorders of platelet function
            - Wiskott-Aldrich syndrome
            - Glanzmann's thrombasthenia
            - Bernard-Soulier syndrome
        - disorders of receptors and signal transduction
            - platelet cyclo-oxygenase deficiency

        - thromboxane synthase deficiency
        - thromboxane A2 receptor defect
        - ADP receptor defect
    - disorders of platelet adhesion
        - von Willebrand's disease
    - disorders of the platelet granules
        - idiopathic dense granule disorder
        - Hermansky-Pudlak syndrome
        - Chediak-Higashi syndrome
        - grey platelet syndrome
        - Paris-Trousseau/Jacobsen's syndrome
    - idiopathic alpha- and dense-granule storage pool disease
    - disorders of phospholipid exposure; Scott syndrome
- acquired platelet function disorders
    - medications and chemicals
        - aspirin
        - other NSAIDs
        - clopidogrel
        - dipyridamole
        - beta-lactam antibiotics
        - dextran
        - alcohol
    - some herbal supplements and foods
        - gingko biloba
        - garlic
        - bilberry
        - ginger
        - ginseng
    - chronic kidney disease
    - heart valve disease, cardiopulmonary bypass, ECMO
    - acquired vWD in patients with aortic stenosis (also reported in Wilm's tumour, hypothyroidism)
    - myeloproliferative disorders
        - e.g. essential thrombocytopenia, polycythaemia vera
    - myelodysplastic syndromes
    - paraproteins
        - especially multiple myeloma and Waldenström's macroglobulinaemia
    - antibody-induced platelet dysfunction
        - bleeding in patients with ITP usually occurs at very low platelet counts

- pseudothrombocytopenia
    - can occur when platelets undergo clumping
        - platelets stick together and give a false low reading when passed through an autoanalyzer
        - caused by the action of EDTA as an anticoagulant

        - occurs in 0.1% of the population but also associated with certain infections, autoimmune disorders, neoplastic disorders, thrombotic disorders and possibly trauma
        - requires examination of a peripheral blood smear
- history
    - epistaxis
        - particularly if excessive, frequent, prolonged
    - bleeding gums or bleeding from tooth extraction
    - haemoptysis, haematemesis, haematuria, haematochezia, melaena
        - can be exacerbated if there is a secondary disorder
    - heavy irregular menstrual bleeding
        - especially in vWD
    - postpartum haemorrhage
    - excessive bleeding during or after surgery (even minor)
    - bleeding after aspirin
    - spontaneous bruising
- examination
    - petechiae (<2mm), purpura (0.2 -1cm) and/or ecchymoses on the skin
    - other abnormalities may provide an indication of an underlying cause
    - it is essential to look for any lymphadenopathy or hepatosplenomegaly
- investigations
    - FBC with repeat and blood smear if platelets are low
    - PT and aPTT
    - renal function
    - TFTs
    - bone marrow examination for patients over 60 and in those with systemic symptoms or signs suggestive of haematological cancer
    - specific assays of inherited platelet dysfunction
- management
    - patients with modest isolated thrombocytopenia without atypical features require safety netting and occasional follow up for monitoring
    - further investigation required for severe thrombocytopenia ($<20 \times 10^9$/L), severe bleeding, red cell fragments or blasts on blood film, constitutional symptoms, bruising, minor bleeding or abnormalities on examination
    - inherited platelet disorders
        - platelet transfusions may be required but there is a risk of alloimmunisation
        - topical measures (TXA soaked gauze, fibrin sealants, splints for dental extractions, packing for nosebleeds)
        - antifibrinolytic agents
        - desmopressin (increases plasma levels of vWF and factor VIII)
        - stem cell or bone marrow transplantation may be considered
        - high doses of oestrogen followed by high doses of oestrogen-progestogen for menorrhagia
        - measures to prevent bleeding including vaccination against hepatitis B, avoidance of NSAIDs, good dental hygiene, correction of iron deficiency

        - acquired platelet disorders
            - specific to the underlying cause
                - may include desmopressin and platelet transfusion
            - antifibrinolytic therapy
                - may be useful, especially for mucosal bleeding
                - should not be used in patients with haematuria or DIC
            - rFVIIa may be used but is associated with increased risk of thrombosis
- aplastic anaemia
    - rare
    - diagnosis requires:
        - Hb <100 g/L
        - platelets <50 $x10^9$/L
        - neutrophils <1.5 $x10^9$/L
    - causes:
        - idiopathic (70-80%)
        - congenital or inherited
        - acquired
            - infection
            - toxic exposure (radiation, chemicals)
            - drugs
                - multiple including sulphonamides, diclofenac, naproxen, phenytoin, carbamazepine, chloroquine
            - graft-vs-host disease
            - pregnancy
            - sickle cell anaemia
    - presentation
        - symptoms of anaemia
            - pallor
            - headache
            - palpitations
            - dyspnoea
            - fatigue
            - ankle oedema
        - symptoms of thrombocytopenia
            - skin or mucosal haemorrhage
            - visual disturbance due to retinal haemorrhage
            - petechial rashes
        - infection
    - investigations
        - FBC, reticulocyte count, blood film
        - HbF in children
        - bone marrow aspirate and trephine biopsy
        - investigation for Fanconi's anaemia if under 35
        - flow cytometry to exclude paroxysmal nocturnal haemoglobinuria
        - vitamin B12 and folate
        - LFTs

        - viral studies
            - hepatitis A, B, C, EBV, CMV
        - antinuclear antibody and anti-dsDNA
    - management
        - course is variable and often mild
        - treatment should be based on degree of cytopenia; asymptomatic patients probably do not need treatment
        - removal of any underlying cause
        - haemopoietic stem cell transplant may be required
        - immunosuppressive therapy
        - supportive care
            - blood transfusions – if on immunosuppressive therapy blood products must be irradiated
            - platelet transfusions I receiving active treatment
            - prophylactic antibiotics and antifungals according to local policy if severely neutropenic
- thrombotic thrombocytopenic purpura
    - background
        - rare life-threatening condition that resembles HUS but can be treated with plasmapheresis
        - autoantibodies against the protease that degrades vWF
            - results in platelet aggregation and clumping
            - there is extensive microthrombi formation with haemolysis and end organ effects
        - may be triggered by an intercurrent event (e.g. surgery, pancreatitis, sepsis, pregnancy) resulting in endothelial activation
    - clinical features
        - fever
        - anaemia
            - microangiopathic haemolytic anaemia
        - thrombocytopenia
        - renal problems
        - neurological problems
            - headaches, confusion, seizures, intracranial haemorrhage, focal deficits
    - risk factors
        - infection
            - enterohaemorrhagic E. coli 0157
        - drugs
            - calcineurin antagonists
            - clopidogrel
            - cyclosporin
            - ticlopidine
        - pregnancy
        - SLE
        - graft versus host disease
        - HIV-1

        - connective tissue disorders
        - malignancy
    - investigations
        - FBC
        - peripheral blood film
        - haemolysis screen
            - reduced haptoglobins
            - increased LDH
            - unconjugated hyperbilirubinaemia
            - increased urinary urobilinogen
            - negative Coomb's test
        - urinalysis
            - near normal
            - exclude active sediment
        - biochemistry
            - increased urea and creatinine levels
        - coagulation
            - normal
        - CT head
            - exclude intracranial pathology if there are seizures
        - other microbiological cultures
        - ADAMTS13 protease
            - low
        - tissue biopsies
    - management
        - plasmapheresis
        - corticosteroids 1mg/kg/day
        - seek and treat thrombotic complications
        - only transfuse if clinically indicated (life-threatening haemorrhage
        - daily platelet levels
        - rituximab IV
- haemolytic uraemic syndrome
    - background
        - clinically similar condition to TTP
        - renal failure more common than neurological symptoms
        - typical and atypical forms
        - can coexist with TTP
    - types
        - diarrhoeal (D+ HUS)
            - >90%
            - caused by preceding infection with a verocytotoxin-producing bacteria, typically E.coli O157:H7
        - non-diarrhoeal/atypical (D- or aHUS)
            - rare
            - possibly familial
            - only about 50% of those with a genetic predisposition develop aHUS
            - poorer prognosis

        - often results from complement dysregulation
    - triggers
        - D+ HUS
            - verocytotoxin-producing bacteria, typically E. coli O157:H7
        - aHUS
            - Streptococcus pneumoniae (40%)
            - pregnancy (term or post-partum)
            - drugs (quinine, mitomycin, ciclosporin, tacrolimus, ticlopidine, caboplatinum, gemcitabine
            - transplantation (renal, haematological stem cell)
            - HIV
            - combined methylmalonicaciduria and homocystinuria (B12 metabolism disorder)
        - P-HUS
            - occurs because Strep. pneumoniae has a neuraminidase that removes N-acetylneuraminic acid from cell-surface glycoproteins
                - exposes the normally hidden T antigen on red blood cells, platelets and glomeruli
            - anti-T immunoglobulin M (IgM) in the serum reacts with the antigen causing haemolysis, thrombocytopenia and renal damage
    - clinical features
        - fever
        - anaemia
        - thrombocytopaenia
        - renal problems
        - neurological features
        - bloody diarrhoea post E. coli infection
    - investigation findings
        - low platelets
        - low Hb with polychromasia, schistocytes and spherocytes
        - increased reticulocytes
        - decreased haptoglobins
        - increased LDH
        - unconjugated hyperbilirubinaemia with urinary urobilinogen
        - variable neutrophilia
        - increased urea and creatinine levels
        - renal biopsy if permitted by platelet levels for confirmation of diagnosis
    - management
        - supportive care and monitoring
            - antibiotics may worsen toxin production
            - platelets are contra-indicated
            - plasmapheresis if unsure
            - seek and treat the underlying cause and any complications
        - specific therapies
            - plasma exchange and plasma infusion
                - uncertain efficacy
            - general treatment of acute renal failure

        - fluid restriction and diuretics
        - anti-hypertensives
        - avoidance of nephrotoxins where possible
        - renal replacement therapy if necessary
- DIC
    - background
        - an acquired syndrome characterised by the intravascular activation of coagulation with loss of localisation arising from different causes
        - can originate from and cause damage to the microvasculature, which can produce organ dysfunction
    - pathophysiology
        - there is an underlying disease process leading to:
            - por-inflammatory cytokines (activation of mononuclear and endothelial cells)
            - intravascular fibrin formation
            - microvascular thrombi and organ dysfunction
            - consumptive intravascular coagulopathy and thrombocytopenia
            - widespread haemorrhage
    - causes include:
        - shock
        - sepsis
        - haemolysis
        - malignancy
            - e.g. promyelocytic leukaemia, other haematological malignancies, solid tumours
        - trauma
            - e.g. multi-trauma, traumatic brain injury, fat embolus
        - pancreatitis
        - severe hepatic failure
        - burns
        - major surgery
        - PE
        - ECMO
        - transplant rejection
        - transfusion reactions
        - obstetric
            - pre-eclampsia
            - amniotic fluid embolism
            - intrauterine death
            - abruption
        - vascular disorder
            - e.g. Kasabach-Merrit syndrome, large aneurysms
    - clinical features
        - may be chronic with few overt clinical effects
        - can be an acute catastrophe
        - haemorrhage
        - microthrombosis leading to multiorgan failure

- investigations
    - anaemia
    - prolonged APTT, INR and PT
    - thrombocytopenia or falling platelets
    - low fibrinogen
    - fragmented RBCs on blood film
    - high fibrin degradation products/d-dimer
    - low levels of plasma coagulation factors and inhibitors
    - DIC score
        - useful for diagnosis
        - simple scoring system based on platelet count, PT, d-dimer and fibrinogen
        - sensitivity 93% and specificity 98%
        - strong independent predictor of mortality in patients with severe sepsis
- differential
    - primary fibrinolysis
    - dilutional coagulopathy from massive transfusion
    - trauma induced coagulopathy
    - post thrombolysis
    - venom induced consumptive coagulopathy (VICC) from snake bite envenoming
- management
    - treat the cause
    - FFP for APTT and INR
    - cryoprecipitate for fibrinogen
    - platelets for thrombocytopenia (aim >50)
    - consider FIIa
    - consider heparin if not bleeding (in chronic DIC)

HP3 massive haemorrhage

- NICE guidelines
    - anticoagulation reversal
        - rapidly reverse anticoagulation
        - do not use plasma to reverse a vitamin K antagonist in patients with major trauma
        - do not reverse anticoagulation in patients who do not have active or suspected bleeding
        - use prothrombin complex concentrate immediately for major trauma with active bleeding requiring emergency reversal of a vitamin K antagonist
        - a haematologist should be consulted for reversal of other agents
    - circulatory access and fluid replacement
        - peripheral IV access or IO access whilst central access is being achieved
        - for patients with active bleeding use a restrictive approach to volume resuscitation until definitive early control of bleeding has been achieved
        - for patients with haemorrhagic shock and a traumatic brain injury consider restrictive volume resuscitation if haemorrhagic shock is the dominant

condition, otherwise use less restrictive volume resuscitation to maintain cerebral perfusion
        - do not use crystalloids for patients with active bleeding
        - use a 1:1 ratio of plasma to red blood cells
    - diagnostic imaging
        - should be performed urgently and the images interpreted immediately by a skilled professional
        - be aware that negative FAST does not exclude intraperitoneal or retroperitoneal haemorrhage
        - consider immediate CT if responding to resuscitation or haemodynamic status normal but suspected to be bleeding
        - FAST should not be used in major trauma before immediate CT or to determine the need for CT
    - dressings and tourniquets
        - use simple dressings with direct pressure to control external haemorrhage
        - in patients with major limb trauma use a tourniquet if direct pressure has failed to control life-threatening bleed
    - haemorrhage protocols
        - use physiological criteria including the patient's haemodynamic status and their response to immediate fluid resuscitation to activate the major haemorrhage protocol
        - do not rely on a haemorrhage risk tool applied at a single point in time
    - haemostatic agents
        - use IV tranexamic acid as soon as possible for patients with major trauma and active or suspected active bleeding
        - do not use tranexamic acid more than 3 hours after injury in patients with major trauma unless there is evidence of hyperfibrinolysis
    - surgery and interventional radiology
        - damage control surgery should be used in patients with haemodynamic instability not responding to volume resuscitation
        - consider definitive surgery in those responding to resuscitation and those with normal haemodynamic status
        - interventional radiology should be used in patients with active arterial pelvic haemorrhage unless immediate open surgery needed for control of bleeding from other injuries
        - consider interventional radiology for patients with solid organ arterial haemorrhage (spleen, liver, kidney)
        - consider joint interventional radiology and surgical approach where arterial haemorrhage extends to surgically inaccessible regions
        - endovascular stent graft should be used in patients with blunt thoracic aortic injury
- major haemorrhage in trauma
    - background
        - find the bleeding, stop the bleeding
        - rapid and effective restoration of blood volume
        - maintain functional blood composition to preserve blood function – haemostasis, oxygen carrying capacity, oncotic pressure and biochemistry
    - source of haemorrhage

        - scalp and external sources
        - chest
        - abdomen
        - long bones
        - pelvis
        - retroperitoneum
        - (consider multiple sources of bleeding, don't stop at the first)
    - approach to haemorrhage control
        - get help early
            - e.g. surgeon, interventional radiology, anaesthetics, ICU
        - find the cause
        - initial measures:
            - direct pressure and elevation
            - adrenaline soaked gauze, haemostatic dressings
            - reduce and splint long bone and pelvic fractures
            - tourniquets
        - invasive measures:
            - sutures
            - tamponade
                - packing or foley catheter with balloon inflated
                - tie off vessels
                - cautery
                - interventional radiology
                - damage control surgery
        - correct coagulopathy
    - pulse palpation as a guide to blood pressure
        - pulses are lost in the order radial, femoral, carotid
        - the SBP at which they disappear varies and traditional ATLS guidance may overestimate it (60-70 carotid, 70-80 femoral, >80 radial)
    - classification of stages of haemorrhagic shock
        - class 1
            - blood loss up to 750ml or 15% blood volume
            - heart rate <100/min
            - blood pressure normal
            - pulse pressure normal/increased
            - respiratory rate 14-20/min
            - urine output >30ml/h
            - CNS – slightly anxious
        - class 2
            - blood loss 750-1500ml or15-30% blood volume
            - heart rate 100-120/min
            - blood pressure normal
            - pulse pressure decreased
            - respiratory rate 20-30/min
            - urine output 20-30ml/h
            - CNS: mildly anxious
        - class 3

                - blood loss 1500-2000ml or30-40% blood volume
                - heart rate 120-240/min
                - blood pressure decreased
                - pulse pressure decreased
                - respiratory rate 30-40/min
                - urine output 5-15ml/h
                - CNS: anxious, confused
            - class 4
                - blood loss >2000ml or >40% blood volume
                - heart rate >140/min
                - blood pressure decreased
                - pulse pressure decreased
                - respiratory rate >35/min
                - urine output negligeable
                - CNS: confused, lethargic
    - problems with the classification of shock
        - differences in compensation for different types of injury (e.g. blunt versus penetrating)
        - age (e.g. blunted physiological responses in the elderly)
        - comorbidities
        - medications (e.g. beta blockers)
        - bradycardia is often seen in major haemorrhage
            - mechanism is not clear
            - some patients seem to have a relative bradycardia and do not mount the initial tachycardia
    - lethal triad and acute coagulopathy of trauma/shock
        - hypothermia
        - coagulopathy
        - acidosis
    - hypothermia prevention and treatment
        - aggressive resuscitation with blood products
        - warmed fluids via fluid warmer
        - Bair hugger or warm blankets
        - minimise exposure
        - increase ambient temperature
        - continuous temperature monitoring
    - damage control resuscitation
        - aims to maintain circulating volume, control haemorrhage and correct the lethal triad until definitive intervention is appropriate
        - three components:
            - permissive hypotension
                - seeks to avoid excessive fluid administration and the associated problems of haemodilution, fluid overload and clot disruption
                - classically advise a target SBP of 80 to 100

        - perfusion is more important than blood pressure so an alternative strategy is to target a MAP >65 mmHg together with a good radial pulse and pulse oximetry waveform
        - if BP is too high, titrated aliquots of fentanyl (e.g. 25 microgram IV) to provide sympatholysis as well as analgesia
    - early haemostatic resuscitation
        - aims to avoid or ameliorate acute coagulopathy of trauma and the complications of aggressive crystalloid fluid resuscitation while maintaining circulating volume
        - involves early use of blood products in ratios resembling that of whole blood
    - damage control surgery
        - limited surgical interventions that serve to control haemorrhage and minimise contamination until the patient has sufficient physiological reserve to undergo definitive interventions
        - aims to bring the lethal triad under control so the patient will be able to tolerate further surgery once they are improving

HC1 anti-coagulant reversal

- general guidance:
    - stop the antithrombotic drug
    - document time and amount of last dose
    - check coag screen, LFTs, U&Es
    - stop source of bleeding if possible
    - IV fluids +/- transfusion as needed
- major life-threatening bleed (including critical area such as intracranial, intraspinal, intra-ocular):
    - warfarin
        - vitamin K (phytomenadione) 5mg IV
            - correction in 6-8h
        - Prothrombin Complex Concentrate - Beriplex/Octaplex (dose 25-50 units/kg)
            - example dosing (see Trust policy):
                - INR 2.0 – 3.9, 25 units/kg
                - INR 4.0-6.0, 35 units/kg
                - INR >6.0, 50 units/kg
            - maximum dose 5000 units
        - consider IV tranexamic acid 1g bolus and 1g TDS
        - FFPs if PCC unavailable
        - repeat INR 30 minutes after PCC/FFP
    - apixaban/rivaraoxaban/edoxaban
        - consider tranexamic acid 1g IV
        - PCCs 50 units/kg (max 5000 units)
    - dabigatran
        - idarucizumab 5g in 100ml infusion over 10-20 minutes
        - consider tranexamic acid

        - PCCs if Praxbind not available
- non-major bleed
    - withhold next dose, consider vitamin K if on warfarin with raised INR

HC2 DIC

- definition:
    - acquired syndrome with intravascular activation of coagulation and loss of localisation
    - generation of fibrin clots can cause organ failure
    - never occurs in isolation, requires management of the underlying condition
        - associated conditions include:
            - sepsis and severe infection
            - trauma
            - organ destruction (e.g. pancreatitis)
            - malignancy
                - solid tumours
                - leukaemia
            - obstetric
                - amniotic fluid embolism
                - placental abruption
                - pre-eclampsia
            - vascular abnormalities
                - large haemangiomata
                - vascular aneurysm
            - severe liver failure
            - toxic and immunological insults
                - snake bites
                - recreational drugs
                - ABO transfusion incompatibility
                - transplant rejection
- diagnosis:
    - no single laboratory test
        - combination of clinical suspicion and tests required
    - platelet count
        - reduction or downward trend sensitive but not specific
    - d-dimer
        - raised, but also raised in many of the contributing conditions
    - PT/aPTT
        - prolonged in 50-60% of cases but may be normal or short
    - fibrinogen
        - can remain in normal range despite ongoing consumption
        - downward trend may be useful
    - ISTH diagnostic scoring system:
        - risk assessment – does the patient have an underlying disorder known to be associated with overt DIC?
            - if yes, proceed; if no, do not use algorithm
        - order global coagulation tests

        - PT, platelet count, fibrinogen, fibrin related marker
    - score the test result
        - platelet count
            - >100 = 0
            - <100 = 1
            - <50 = 2
        - fibrin marker (e.g. d-dimer)
            - no increase = 0
            - moderate increase = 2
            - strong increase = 3
        - prolonged PT
            - <3s = 0
            - >3 but <6s = 1
            - >6s = 2
        - fibrinogen level
            - >1 =0
            - <1 = 1
    - calculate score
        - ≥5 compatible with overt DIC – repeat score daily
        - <5 suggestive for non-overt DIC – repeat score next 1-2 days
- management:
    - treat the underlying condition
    - consider platelet transfusion if count <50 and invasive procedure required
    - consider FFP if prolonged PT and aPTT and bleeding
    - VTE prophylaxis
    - if thrombosis predominates, consider therapeutic doses of heparin or infusion if high bleeding risk

HC3 haemophilia

- haemophilia A
    - factor VIII deficiency
    - usually inherited, rarely acquired
    - severity depends on remaining factor activity
    - cause:
        - mutations in factor VIII gene that map to Xq28
        - usually X-linked recessive (males born to carrier mothers)
        - usually clear family history
        - rarely, females born to affected fathers can have the disease due to homozygosity
    - epidemiology
        - 1:4,000-1:5,000 male births worldwide
        - five times more common than haemophilia B
    - presentation:
        - spontaneous bleeding into joints, especially knees, ankles and elbows without history of significant trauma
        - haematuria may occur
        - GI and mucosal bleeding less common

        - untreated severe cases may develop:
            - arthropathy and joint deformity
            - soft tissue haemorrhage (including compartment syndrome and neurological damage)
            - extensive retroperitoneal bleed
            - haematoma formation
        - moderate disease often presents with bleeding after venepuncture
        - mild disease only has bleeding after major trauma or surgery
    - management:
        - prophylaxis
            - infusions of factor VIII
            - tailored to individual (e.g. before physical activity)
            - before occurrence of a second joint bleed or significant soft tissue bleed
            - continue until physical maturity
                - can be reinstated after this if indicated
        - acute bleeding episodes
            - patients should administer own normal factor VIII
            - in-hospital options:
                - recombinant factor VIII (preferred)
                - monoclonal antibody purified factor VIII
                - FFP containing factor VIII (may cause antibodies to the deficient protein and complicate future treatment)
            - aim to correct factor VIII activity to 100% for severe haemorrhage (CNS, GI, GU, retroperitoneal, trauma, severe epistaxis) and 30-50% for minor haemorrhage (haemarthrosis, oral mucosal, muscular)
            - enhanced levels are maintained for 7-10 days for severe bleeds and 1-3 days for minor bleeds
            - desmopressin (DDAVP) and antifibrinolytic agents may be used to boost factor VIII activity and reduce administration requirements
            - review prophylaxis regimen after resolution
- haemophilia B (Christmas disease)
    - factor IX deficiency
    - x-linked recessive
    - tends to be less severe than haemophilia A
    - the lower the level of factor IX, the more severe the disease
    - presentation:
        - spontaneous haemorrhage and haemarthroses in severe disease
        - haemorrhage from minor trauma or surgery, sometimes spontaneous haemarthrosis in moderate disease
        - unexpected haemorrhage after trauma or surgery or precipitated by NSAIDs in mild disease
    - management:
        - prophylaxis with factor IX therapy
            - risk of inhibitors less than with factor A
            - can present as anaphylaxis
        - acute

- self-administered concentrate if possible
- coag and G&S
- recombinant factor IX if available
    - correct to 100% for serious haemorrhage, 50% for minor haemorrhage
    - further doses will be required
    - FFP and cryoprecipitate may be used if needed
- avoid NSAIDs
- opiates can be given IV or SC but not IM (risk of large haematoma)
- give tranexamic acid for oral mucosal bleeds

- von Willebrand disease
    - deficiency or abnormal function of von Willebrand factor (vWF)
        - vWF assists in platelet plug formation by attracting platelets to the site of the damage and binds to factor VIII preventing clearance from the plasma
    - prevalence 1-2% in general population
    - most have mild disease
    - more common in females
    - more severe with blood type O
    - types:
        - hereditary
            - type 1
                - 60-80%
                - quantitative defect
                - autosomal dominant
                - easy bruising and/or menorrhagia, bleeding after dental work/major surgery
            - type 2
                - 20-30%
                - qualitative defect
                - usually autosomal dominant, can be autosomal recessive
                - variable bleeding tendency
            - type 3
                - 1-5%
                - most severe type
                - quantitative
                - autosomal recessive
                - low factor VIII
                - severe mucosal bleeding, may have haemarthrosis
        - acquired
            - pseudo-von Willebrand's disease
            - found in lymphoproliferative or myeloproliferative disorders
            - can be associated with solid tumours
            - also with other conditions (e.g. aortic stenosis, Wilm's tumour, hypothyroidism)
    - presentation:
        - bleeding from mucosa
            - epistaxis, menorrhagia

        - spontaneous bleeding (severe cases)
            - internal or joint bleeding
        - blood clots during childbirth
    - management:
        - often no specific treatment required
        - options are:
            - tranexamic acid
                - topical, mouthwash, oral, IV
            - desmopressin
                - temporarily increases factor VIII and vWF levels by releasing endothelial stores
                - intranasal or parenteral
            - concentrates of high-purity vWF or intermediate purity factor VIII-vWF
            - platelet transfusions may be helpful

HC4 ITP

- idiopathic thrombocytpaenic purpura/immune thrombocytopaenic purpura
    - autoimmune haematological disorder
    - isolated thrombocytopaenia in absence of identifiable cause
        - platelets <100
        - normal platelets last 8-10 days but are destroyed in hours in ITP
- presentation:
    - typically in children
        - peak incidence 2-5 years
        - often with preceding viral illness or immunisation
    - more common in females in adults
    - petechiae (1-5mm non-blanching spots)
        - primarily on lower limbs but can be anywhere
    - mucocutaneous bleeding
        - rarely severe
    - no other symptoms or autoimmune disorders, normal examination with no hepatosplenomegaly or lymphadenopathy
- diagnosis:
    - low platelet count with normal haemoglobin
- management:
    - based on symptoms not platelet count
    - assess for complications:
        - haematuria
        - melaena
        - menorrhagia
        - epistaxis
        - mucosal bleeding
        - tonsillar purpura/petechiae
    - steroids
        - prednisolone 1-2mg/kg OD for 3 weeks then taper
        - or methylprednisolone 30mg/kg for 3 days then 20mg/kg for four days

    - IVIG
        - if significant bleeding
        - works in 1-5 days to raise platelet count and lasts 2-4 weeks
    - platelets
        - only if intracranial haemorrhage or significant bleeding
        - IVIG given first prolongs platelet survival
- admission criteria:
    - significant bleeding
        - epistaxis >1 hour
        - haematemesis
        - haemoptysis
        - intracranial haemorrhage
        - melaena
    - unclear diagnosis
    - problematic social circumstances
- prognosis:
    - 80% resolves by 6 months (with or without treatment)
    - 5% have a recurrence
    - 10% have chronic ITP
    - more likely to be chronic in adults
- safety-netting:
    - inform parents to look out for signs of intracranial haemorrhage, urinary bleeding, GI bleeding, excessive mucosal bleeding. menorrhagia
    - avoid NSAIDs whilst platelet count low
    - avoid contact sports in older children (impractical for younger children)
- follow up:
    - review within 2 weeks of initial presentation with repeat FBC
    - weekly GP follow up initially then as needed
    - paeds OP review at 6 weeks, 3 months and 6 months

HC5 leukaemia

- acute myeloid leukaemia (AML)
    - maturational arrest of bone marrow cells in first stages of development
        - reduces number of normal blood cells
    - failure of apoptosis leads to accumulation in various organs
    - 8 different subtypes under FAB (French-American-British) classification system
    - epidemiology:
        - most common acute leukaemia in adults
        - European incidence 5-8 cases per 100,000
        - incidence increases with age, median 67
    - predisposing factors:
        - MDS
        - aplastic anaemia
        - myelofibrosis
        - paroxysmal nocturnal haemoglobinuria
        - polycythaemia rubra vera
        - CML

        - certain congenital disorders including Down's syndrome, Fanconi's anaemia and neurofibromatosis
        - previous chemotherapy
    - presentation:
        - more acute in younger patients, older patients may have fatigue over weeks or months
        - dizziness and shortness of breath on exertion
        - very high WCC and low neutrophils
        - fever
        - bleeding
        - splenomegaly
            - fullness in LUQ, early satiety
        - leukostasis
            - secondary to WCC >100
            - respiratory distress and altered mental status
            - medical emergency
        - bone pain
        - on examination may have hepatosplenomegaly, thrombocytopaenia, gingivitis, pallor
    - investigation:
        - FBC
        - coag
        - LDH
        - uric acid
        - LFTs/U&Es
    - management:
        - usually two phases of induction and intensification with chemotherapy
        - stem cell transplant
        - other management includes antibiotics for infection, blood product replacement, allopurinol to reduce uric acid levels
    - prognosis:
        - 80% remission in children
        - 80% remission in younger adults
        - 60% remission in over 60s but usually transient, median survival 5-10 months
- acute lymphoblastic leukaemia (ALL)
    - malignant transformation of a clone of cells from lymphoid progenitor cells
        - usually of B cell origin
        - lymphoid precursors proliferate and replace normal bone marrow cells, blasts enter peripheral circulation
    - epidemiology:
        - most common childhood cancer
        - incidence 3 per 100,000
        - peak incidence 2-4 years, small peak in over 50s
        - likely has genetic and environmental factors (including radiation exposure); possible relation to lack of exposure to infection early in life
    - presentation:
        - initial presentation with generalised fatigue and malaise

        - palpitations
        - severe and unusual joint pain
        - recurrent and severe infections
        - fever without obvious infection
        - LUQ fullness and early satiety (splenomegaly)
        - dyspnoea
        - headache, irritability, altered mental state, neck stiffness
        - bleeding and bruising
        - signs include:
            - pallor
            - tachycardia and flow murmur
            - petechiae
            - abdominal distension with hepatosplenomegaly
            - lymphadenopathy
            - testicular enlargement
            - gum hypertrophy
            - leukaemia cutis
            - cranial nerve palsy
    - management:
        - usually remission induction, intensification and maintenance chemotherapy plus supportive measures
    - prognosis:
        - very poor prognosis if relapse
        - 80-90% cure rate in children
        - <10% cure rate in elderly/frail patients
- chronic myeloid leukaemia (CML)
    - myeloproliferative disorder of pluripotent haematopoietic stem cells affecting one or all cell lines (erhythroid, platelet, myeloid)
    - more than 90% associated with Philadelphia chromosome
    - phases:
        - chronic
            - competent immune system, asymptomatic
            - >90% are diagnosed in the initial chronic phase
        - accelerated
            - 15-29% blasts in blood or bone maroow, >20% basophils in blood, thrombocytosis, thrombocytopenia, clonal chromosomal abnormalities
            - features include resistance to therapy, increased constitutional symptoms, progressive splenomegaly
        - blast crisis/blastic phase
            - 1/3 move directly from chronic to blastic stage
            - aggressive acute leukaemia, highly refractory to therapy, usually rapidly fatal
            - severe constitutional symptoms (weight loss, fever, night sweats, bone pain), infection, bleeding

- epidemiology:
  - 1-2 cases per 100,000/year
  - unknown aetiology
- presentation:
  - 40% diagnosed before any symptoms occur
  - symptoms
    - fatigue
    - night sweats
    - weight loss
    - abdominal fullness or distension
    - LUQ pain due to splenic infarction
  - signs
    - splenomegaly
    - hepatomegaly
    - lymphadenopathy
    - hyperdynamic circulation secondary to anaemia
    - easy bruising
    - fever
    - gout
    - hyperviscosity syndrome due to leukocytosis – visual disturbance, priapism, CVA, confusion
- management:
  - 3 types of remission
    - haematologic
      - normal FBC, physical examination
    - cytogenic
      - normal chromosome returns with 0% Ph-positive cells
    - molecular
      - negative PCR for mutational BCR-ABL mRNA
  - drug therapy usually with tyrosine kinase inhibitors (e.g. imatinib)
  - stem cell transplant can be considered but carries risks (graft-versus-host disease, veno-occlusive disease, life-threatening infections, risk of secondary malignancy, poorer overall quality of life)
    - ideally done in chronic phase
- prognosis:
  - five year survival rate on imatibib of 89%

- chronic lymphocytic leukaemia (CLL)
  - malignant monoclonal expansion of B lymphocytes with accumulation of abnormal lymphocytes in blood, bone marrow, spleen, lymph nodes and liver
    - lymphocytes have normal appearance but are immature and non-reactive
  - epidemiology:
    - most common leukaemia in West
    - 4.2 per 100,000/year, increasing to >30 per 100,000/year at age 80
    - median diagnosis 72 years
    - strong familial basis
  - presentation:
    - variable, usually asymptomatic at presentation

        - symptoms:
            - susceptibility to infection
            - symmetrically enlarged lymph nodes
            - abdominal discomfort from enlarged spleen
            - bleeding/petechiae
            - fatigue
        - signs:
            - local or generalised lymphadenopathy
            - hepatosplenomegaly
            - petechiae
            - pallor
            - skin infiltration
            - tonsillar enlargement
            - rare involvement of lacrimal and salivary glands
    - staging (Binet system):
        - stage A
            - Hb at least 100, platelets at least 100, fewer than 3 lymph node areas
        - stage B
            - Hb and platelets as stage A, 3 or more lymph node areas involved
        - stage C
            - Hb <100, platelets <100 or both
    - management:
        - no curative treatment
        - chemotherapy for patients with active, symptomatic disease
            - alkylating agents, purine analogues, +/- monoclonal antibodies
        - steroids
            - to treat autoimmune complications, improve bone marrow function prior to chemotherapy or treat CLL not responding well to chemotherapy
        - allogenic stem ell transplant is potentially curative but risks are rarely justified in older age group; may be attempted in younger patients
        - splenectomy may be required for splenomegaly and pancytopaenia
        - radiotherapy for palliation (spleen or bulky nodal masses)
    - prognosis:
        - usually has long overall survival
        - median survival 18 months for stage C and >10 years for stage A

HC6 lymphoma

- Hodgkin's lymphoma
    - malignant tumour of lymphatic cells
        - histologically has presence of multinucleated giant cells (Reed-Sternberg cells)
            - associated abnormal and smaller mononuclear cells originating from B lymphocytes
    - can be classical or nodular lymphocyte predominant
    - epidemiology:

        - UK incidence 2.7 per 100,000 per year
        - slight male predominance
        - peak incidence 20-34 years, further peak over 70 years
    - risk factors:
        - previous EBV
        - HIV
        - immunosuppression
        - cigarette smoking
    - presentation:
        - enlarged lymph node, typically in lower neck of supraclavicular region
        - mediastinal mass (may be found on routine CXR)
        - chest discomfort, cough, dyspnoea
        - systemic symptoms (B symptoms)
            - drenching night sweats
            - unexplained fever
            - weight loss >10% over 6 months
        - alcohol induced pain at sites of nodal disease
        - signs include:
            - lymphadenopathy
            - hepatosplenomegaly
            - superior vena cava syndrome
            - features of paraneoplastic syndrome
    - investigations:
        - FBC
        - ESR
        - monospot
        - LFTs
        - HIV
        - lymph node biopsy
        - CXR
        - CT TAP for staging
    - staging (Ann Arbor system)
        - stage I: one lymph node region or lymphoid structure (e.g. spleen, thymus, Waldeyer's ring)
        - stage II: 2 or more lymph node regions on same side of diaphragm
        - stage III: lymph nodes on both sides of diaphragm
- splenic, hilar, coeliac or portal nodes
- para-aortic, iliac or mesenteric nodes
        - stage IV; involvement of extranodal sites beyond E
    - modifying features:
        - A: no symptoms
        - B: fever, drenching night sweats, weight loss >10% in 6 months
        - X: bulky disease, >1/3 widening of mediastinum or >10cm maximum diameter of nodal mass
        - E: involvement of single, contiguous or proximal extranodal site
    - limited, intermediate or advanced:
        - limited: up to IIB with no risk factors

        - intermediate: up to IIB with at least 3 lymph node areas or ESR >50 without B symptoms or over 30 with B symptoms
        - advanced: stage IIB with large mediastinal mass or extranodal disease; any stage III or above
    - management:
        - early stage disease treated with combination chemotherapy and involved field radiation therapy, advanced stage disease has a longer course of chemotherapy often without radiotherapy
            - chemotherapy carries increased risk of leukaemia
        - relapse usually managed with high dose chemotherapy followed by autologous stem cell transplant
    - prognosis:
        - localised and advanced can be cured in most patients (80-90%)
        - poor prognostic indicators:
            - increasing tumour burden and extension of disease
            - increasing age
            - male
            - constitutional symptoms
            - anaemia, leukocytosis, lymphopenia, monocytosis
            - low albumin
            - increasing ESR
            - elevated beta-2 microglobulin
            - biohumoral factors
            - circulating DNA of cellular and viral (EBV) origin
            - genetic backgroun
- Non-Hodgkin's lymphoma
    - heterogenous group of lymphoproliferative malignancies
        - more likely to disseminate to extranodal sites than Hodgkin's lymphoma
    - two prognostic groups:
        - low-grade
            - relatively good prognosis, median survival 10 years
            - usually not curable in advanced stages
        - high-grade
            - shorter natural history
            - significant number can be cured with intensive treatment
    - classification:
        - WHO Classification (update of Revised European American Lymphoma Classification) includes:
            - precursor B-cell neoplasm
            - mature (peripheral) B-cell neoplasm
                - high-grade
                    - diffuse large B-cell lymphoma (most common)
                    - mediastinal large B cell
                    - primary central nervous system lymphoma
                    - primary effusion lymphoma
                    - Burkitt's lymphoma
                    - mantle cell lymphoma

        - low-grade
            - follicular lymphoma
            - mucosa-associated lymphoid tissue (MALT) lymphoma
            - Waldenström's macroglobulinaemia
    - precursor T-cell neoplasm
    - mature (peripheral) T-cell neoplasm
        - high-grade
            - enteropathy-type T-cell lymphoma
            - peripheral T-cell lymphoma
            - subcutaneous panniculitis-like
            - systemic anaplastic
            - angio-immunoblastic
        - low-grade
            - mycosis fungoides and cutaneous T-cell lymphomas
- epidemiology:
    - more than five times as common as Hodgkin's disease
    - higher risk in white people
    - median age older than 50
- risk factors:
    - chromosomal translocations and molecular rearrangements
    - viruses (e.g. EBV, human T-cell leukaemia virus type 1, hepatitis C, Kaposi's sarcoma-associated herpesvirus)
    - environmental factors (e.g. pesticides, solvents, dusts, hair dye, chemotherapy, radiation)
    - congenital and acquired immunodeficiency
    - autoimmune disorders
    - H. pylori
- presentation:
    - low-grade
        - painless, slowly progressive peripheral lymphadenopathy
        - primary extranodal involvement and systemic symptoms (fatigue, weakness, fever, night sweats, weight loss) are common in advanced/end stage disease
        - hepatosplenomegaly
    - intermediate and high-grade
        - rapidly growing and bulky lymphadenopathy
        - systemic symptoms and extranodal involvement more common
        - hepatosplenomegaly
        - obstructive hydronephrosis secondary to bulky retroperitoneal lymph nodes
        - testicular mass
        - skin lesions
- staging (Ann Arbor)
    - stage I: single lymph node region or localised involvement of a single extralymphatic organ or site (IE)

        - stage II: 2 or more lymph node regions on same side of diaphragm, or localised involvement of single associated extralymphatic organ in addition to criteria for stage 2 (IIE)
        - stage III: lymph node regions on both sides of diaphragm, may be accompanied by localised involvement of an extralymphatic organ or site (IIIE), spleen (IIIS) or both (IIISE)
        - stage IV: disseminated or multifocal involvement of one or more extralymphatic sites with or without associated lymph node involvement or isolated extralymphatic organ involvement with distant node involvement
        - letters represent extralymphatic organs: L-lung, H-liver, P-pleura, O-bone, M-bone marrow, D-skin
    - management:
        - variable treatment options
            - watchful waiting, single-agent or multi-agent chemotherapy, regional or extended radiotherapy
        - surgery in some situations
        - vaccinations (polyvalent pneumococcal and influenza)
        - monoclonal antibodies

HC7 marrow failure

- bone marrow
    - consists of red marrow and yellow marrow (inactive adipose tissue)
    - mainly found in pelvis, ribs and ends of long bones
    - has islands of erythropoietic cells, some of which are pluripotent stem cells
- bone marrow failure
    - can affect RBCs, WBCs and platelets with single line deficiency or pancytopaenia
    - inherited or acquired
        - inherited
            - Fanconi's anaemia
            - Diamond-Blackfan anaemia
            - dyskeratosis congenita
            - Schwachman-Diamond syndrome
            - congenital amegakaryocytic thrombocytopaenia
            - reticular dysgenesis
        - acquired
            - autoimmunity (most cases of aplastic anaemia)
            - antineoplastic agents and other pharmacological agents (e.g. steroids, NSAIDs, allopurinol, anti-thyroid medication, chloramphenicol, gold), poisons (e.g. benzene)
            - malignancy causing bone marrow infiltration (e.g. lymphoma, multiple myeloma, carcinoma, hairy cell leukaemia)
            - myelodysplasia
            - ionising radiation
            - viruses (hepatitis B, EBV, parvovirus B19)
            - paroxysmal nocturnal haemoglobinuria
            - vitamin B12 or folate deficiency
    - presentation:

        - anaemia
            - tiredness, weakness, pallor, breathlessness, tachycardia
        - neutropaenia
            - recurrent or severe bacterial infections
        - thrombocytopaenia
            - easy bruising, petechiae, bleeding from nose/gums
        - other clinical features associated with underlying cause
    - management:
        - depends on underlying cause
        - transplant
            - definitive treatment for inherited marrow failure and some acquired causes
        - transfusions
        - antibiotics
            - febrile neutropaenia is a medical emergency
        - immunosuppression
            - where transplant not an option

HC8 sickle cell disease/crisis

- caused by HbS haemoglobinopathy
    - produces rigid, distorted, dysfunctional erythrocytes called sickle cells
- precipitants:
    - infection
    - dehydration
    - hypoxia
    - drugs (sedatives, local anaesthetics)
- risk of infection due to immunosuppression
- presentation:
    - fever
        - veno-occlusive disease
        - acute chest syndrome
        - osteomyelitis
        - local or systemic infection
    - vaso-occlusive crisis
        - assume cause of any painful presentations
    - acute chest syndrome
        - life-threatening lung infarction
        - assume if hypoxia and chest pain
    - acute splenic sequestration
        - typically in infants
        - increased spleen size
        - fall in Hb of >20g/L, thrombocytopaenia, normal or increased reticulocytes
    - aplastic crisis
        - fall in Hb and reticulocytes <1%
    - stroke
    - priapism
        - stuttering (2-4h and may be recurrent)

        - severe (>4h, may lead to permanent impotence)
- investigation:
    - FBC and film
    - CXR if fever, chest pain, hypoxia
    - CT head if suspected stroke
- management:
    - correct hypoxia
    - adequate hydration
    - transfuse for symptomatic anaemia, target Hb >50
        - avoid over-transfusion, risk of hyperviscosity or rebound if splenic sequestration
    - fever
        - empiric flucloxacillin and gentamicin
    - vaso-occlusive crisis
        - aggressive pain management (paracetamol +/- NSAIDs, opiates
        - rehydration with oral or IV fluids
    - acute chest syndrome
        - O2 and aggressive pain management
        - consider empiric antibiotics
        - consider plasmapheresis
    - acute splenic sequestration
        - oxygen, hydration, RBC transfusion
    - aplastic crisis
        - oxygen, hydration, RBC transfusion
    - stroke
        - avoid thrombolytics, avoid hyperviscosity
    - priapism
        - oxygen, rehydration, analgesia
        - may need penile aspiration and washout under urology
        - may need exchange transfusion under haematology

HC9 transfusion reactions

- early:
    - TACO (transfusion associated circulatory overload)
        - acute or worsening respiratory compromise and/or evidence of pulmonary oedema during or up to 12 hours after transfusion and 3 or more criteria:
            - required
                - A – acute or worsening respiratory compromise
                - B – acute or worsening pulmonary oedema based on physical examination and/or CXR
            - additional criteria
                - C – evidence for cardiovascular system changes (e.g. tachycardia, hypertension, widened pulse pressure)
                - D – evidence of fluid overload (e.g. positive fluid balance, response to diuretics)
                - E – supportive result of relevant biomarker such as elevated BNP

        - increased risk in patients over 60, infants and the severely anaemic
        - monitoring is key to recognition
        - frequently confused with TRALI (can co-exist); hypertension is constant in TACO but infrequent/transient in TRALI
        - management:
            - stop transfusion
            - upright position
            - oxygen
            - diuretics
            - consider intubation and ventilation
            - report to blood bank and haematology
    - TRALI (transfusion related acute lung injury)
        - hypoxia and pulmonary oedema during or within 6 hours of transfusion in absence of other causes
        - incidence 1 in 5,000 units
        - likely to have priming of lung capillaries buy underlying illness then trigger of exposure to irritant in blood transfusion
        - clinical features:
            - dyspnoea
            - hypoxia
            - fever
            - hypotension or hypertension
            - acute onset
            - bilateral pulmonary infiltrates
        - differential includes: acute pulmonary oedema, aspiration pneumonitis, inhalational injury, ARDS, fat embolism, diffuse alveolar haemorrhage
        - management:
            - stop transfusion
            - respiratory support (may need NIV or intubation)
                - lung protective ventilation if intubated
            - haemodynamic support if needed
            - inform blood bank and haematology
        - prognosis:
            - most recover within 48-96 hours
            - radiological changes last 7 days
            - mortality 5%
    - haemolytic reactions (incompatibility – ABO, Rh, Kidd)
    - fever
    - allergy (mild to anaphylaxis)
    - infection – bacterial contamination
    - air embolism
    - hypothermia
- late
    - viral infection (hepatitis B, HIV, CMV)
    - bacterial infection (Treponema pallidum, Salmonella, Yersinia, Pseudomonas, Staphylococcus spp)
    - parasitic infection (malaria, toxoplasmosis)

- prion infection
- graft versus host disease
    - most common in immunocompromised patients
    - generally devastating and fatal
    - onset 2-4 weeks after transfusion
    - features:
        - fever
        - abnormal LFTs
        - profuse watery diarrhoea
        - erythematous skin rash
        - progressive marrow failure
- immune sensitisation (Rh D antigen)
- TRIM (transfusion-related immunomodulation) – leading to risk of infection, tumour, recurrence, activation of latent viral infections, recurrent miscarriages

INFECTIOUS DISEASES

IP1 fever

- background
    - defined as temperature ≥38°
    - peripheral temperature is not clinically accurate, central measurements are preferred (rectal or oral)
        - rectal temperatures should not be performed in neutropenic patients
    - fever causes an increase in heart rate (10bpm per degree) and respiratory rate
- differential
    - infectious
        - critical
            - sepsis
            - pneumonia with respiratory failure
            - peritonitis
            - meningitis
            - cavernous sinus thrombosis
            - necrotising fasciitis
        - emergent
            - pneumonia
            - peritonsillar abscess
            - retropharyngeal abscess
            - epiglottitis
            - endocarditis
            - pericarditis
            - appendicitis
            - cholecystitis
            - diverticulitis
            - intra-abdominal abscess
            - pyelonephritis

- tubo-ovarian abscess
- encephalitis
- brain abscess
- cellulitis
- abscess
- malaria

- non-emergent
  - viral syndrome
  - otitis media
  - sinusitis
  - pharyngitis
  - influenza
  - TB
  - UTI
  - epididymitis
  - prostatitis

- non-infectious
  - critical
    - myocardial infarction
    - PE
    - intracranial haemorrhage
    - CVA
    - neuroleptic malignant syndrome
    - thyroid storm
    - acute adrenal insufficiency
    - transfusion effects
    - pulmonary oedema
    - heat stroke
    - malignant hyperthermia
  - emergent
    - congestive heart failure
    - dehydration
    - recent seizure
    - sickle cell disease
    - transplant rejection
    - pancreatitis
    - DVT
    - serotonin syndrome
  - non-emergent
    - drug fever (not neuroleptic malignant syndrome or serotonin syndrome)
    - malignancy
    - gout
    - sarcoidosis
    - Crohn's disease
    - post myocardiotomy syndrome

- Sweet's syndrome (acute febrile neutrophilic dermatosis)
- management
  - treat the underlying cause as needed
  - symptomatic relief with antipyretics

IP2 pyrexia in travellers

- history
  - symptoms
    - nature
    - onset
    - duration
  - travel departure and return dates
  - area of travel
  - nature of terrain
    - rural, urban, forest, mountain
  - duration of visit
    - risk increases with length of stay
  - purpose of travel
  - behaviour and lifestyle of traveller
  - degree of contact with local population
  - known disease contacts
  - known insect or animal bites, scratches or licks
  - unprotected intercourse
  - diet whilst travelling
  - vaccination history
  - malaria prophylaxis and compliance
  - injuries or illnesses
    - how and when treated
    - blood taken
    - injections given
    - blood transfused
    - surgery
    - sterility of equipment and supplies
- findings needing urgent investigation
  - rash
  - breathing difficulty
  - altered conscious level
  - bruising or unusual bleeding
  - persistent vomiting
  - paralysis
  - jaundice
- causes of tropically acquired fever by incubation period
  - short (<10 days)
    - arboviral infections
      - dengue
      - chikungunya
    - gastroenteritis (bacterial or viral)

        - relapsing fever (borrelia)
        - respiratory infection (bacterial or viral)
        - rickettsial infection
            - e.g. typhus, Rocky Mountain spotted fever
        - malaria
        - Lassa fever/Marburg virus
        - Ebola virus (2-12 days)
        - West Nile virus
        - cholera
        - plague
        - yellow fever (3-16 days)
        - influenza
        - SARS
    - medium (10-21 days)
        - bacterial
            - brucellosis
            - enteric fever
                - typhoid and paratyphoid
            - leptospirosis
            - meliodosis
            - Q fever (Coxiella burnetii)
        - fungal/parasitic
            - coccidioidomycosis
            - histoplasmosis
            - acute Chagas disease
            - malaria
            - trypanosoma brucei rhodesiense (sleeping sickness)
        - viral
            - Ebola virus
            - CMV
            - EBV
            - HIV
            - viral haemorrhagic fever
            - yellow fever
    - long (>21 days)
        - bacterial
            - brucellosis
            - TB
        - fluke
            - acute schistosomiasis
        - protozoal
            - amoebic liver abscess
            - malaria
            - trypanosoma brucei rhodesiense
            - visceral leishmaniasis
        - viral
            - HIV

- hepatitis
- rabies

IP3 sepsis

- background
  - sepsis is life-threatening organ dysfunction due to a dysregulated host response to infection
  - septic shock is a subset in which underlying circulatory and cellular/metabolic abnormalities are profound enough to substantially increase mortality
  - SIRS definition
    - temperature >38 or <36
    - HR >90
    - RR >20
    - WCC >12
  - sepsis:
    - SIRS + confirmed or presumed infection
    - mortality 10-15%
  - severe sepsis
    - sepsis with organ dysfunction
    - includes:
      - SBP <90 or MAP <65 or lactate >2 after initial fluid challenge
      - INR >1.5
      - bilirubin >34
      - urine output <0.5 ml/kg/hr for 2 hours
      - creatinine >177
      - platelets <100
      - sats <90% on air
    - mortality 17-20%
  - septic shock
    - sepsis with refractory hypotension
      - SBP <90 or MAP <70
      - persisting after 30ml/kg crystalloid (i.e. vasopressor dependency after adequate volume resuscitation)
    - mortality 43-54%
- risk factors
  - extremes of age
  - instrumentation or surgery
  - indwelling line or catheter
  - alcohol misuse
  - diabetes mellitus
  - breach of skin integrity (e.g. burns)
  - immunocompromise
  - medications (e.g. corticosteroids, chemotherapy)
  - intravenous drug misuse
  - pregnancy
- pathophysiology
  - organisms

        - bacteria
        - fungi
        - viruses
        - parasites
    - complex interaction between:
        - inciting microbe
        - host immune response
        - inflammatory pathway
        - coagulation pathway
    - pro-inflammatory mediators and pathways
        - cytokines
            - TNF, IL-1, IL-6, IL-8, IFN-y
        - coagulation pathways
        - macrophages, monocytes, neutrophils
        - endothelial cells
        - platelets
        - oxygen free radicals
        - proteases
        - NO
    - anti-inflammatory mediators
        - IL-4, IL-10, IL-11, IL-13
        - transforming growth factor beta
        - CSF
        - soluble TNF receptors
        - IL-1 receptor antagonist
        - natural anticoagulants
    - lactic acidosis
        - impaired regional microvascular blood flow and autoregulation
        - mitochondrial dysfunction with impaired pyruvate oxidation
        - excess catecholamines may impair hepatic lactate extraction by reducing regional hepatic blood flow
        - lactate clearance is decreased because pyruvate dehydrogenase activity is reduced in both skeletal muscle and liver
- initial management
    - resuscitation
        - consider blood transfusion if cleeding or anaemic
            - target Hb >70 in non-bleeding patients
    - early administration of appropriate antibiotics following blood cultures
        - as early as possible and within one hour of arrival
        - 2 sets of blood cultures should be taken prior to antibiotic administration
    - early source control
        - imaging as needed
        - operative interventions such as laparotomy, incision and drainage of abscesses
        - consider removal of pre-existing in situ devices
    - judicious fluid resuscitation, avoiding excess fluids
        - most patients need no more than 2-3 litres (30ml/kg)
        - use 0.9% sodium chloride or Hartmann's

    - noradrenaline for refractory hypotension
        - can be given peripherally in the first 6 hours with close observation for extravasation
        - maintains coronary perfusion by increasing diastolic blood pressure through systemic arterial vasoconstriction
        - increases preload by venoconstriction
        - adrenaline is an acceptable alternative
        - phenylephrine and metaraminol are not suggested
    - inotropes for septic cardiomyopathy
        - generally dobutamine
    - therapies for refractory hypotension
        - target MAP >65 in most patients
        - consider adding vasopressin
    - ongoing supportive care and monitoring
- sepsis six
    - administer high flow oxygen
    - take blood cultures and consider source
    - administer IV antibiotics
    - give IV fluid resuscitation
    - check Hb and serial lactates
    - commence hourly urine output measurement

IP4 needlestick injury/exposure to blood borne viruses

- definition
    - wound piercing the skin caused by a contaminated sharps instrument, usually a hypodermic needle
    - an occupational exposure occurs when a healthcare worker is exposed to blood or bodily fluids of another person
    - most significant risk is transmission of blood borne viruses (HIV, hepatitis B, hepatitis C)
- context
    - occupational hazard for healthcare workers
    - most common among nursing staff - 48% have one at some point in their career
    - the ED should provide initial assessment and management for healthcare workers if occupational health not available
    - members of the public may also present with needlestick injuries
    - significant infective consequences are rare
        - between 1997 and 2018 in the UK:
            - 23 cases of seroconversion to active infection of hepatitis C, all through hollow bore needles
            - 1 case of HIV seroconversion in 1999
            - no reported cases of hepatitis B seroconversion
    - risk of transmission of BBV:
        - hepatitis B
            - UK prevalence <1%
            - risk of transmission 1:3
        - hepatitis C

        - UK prevalence <0.5%
        - risk of transmission 1:30
    - HIV
        - UK prevalence <0.3
        - risk of transmission 1:300
- risk assessment for healthcare workers
    - whether the fluid involved was significant
        - significant fluids:
            - blood
            - internal body fluids
            - semen
            - vaginal secretions
            - saliva
        - non-significant
            - urine
            - faeces
    - whether the nature of the exposure was significant
        - significant incidents include:
            - percutaneous injury from anything that breaks the skin e.g. needles, instruments, bone fragments, bites
            - splash exposure to broken skin, abrasions, wounds, eczema
            - splash exposure to mucous membranes, most commonly the eye
        - four factors are associated with increased risk of occupationally acquired HIV infection after needlestick injury:
            - deep injury
            - visible blood on the device involved
            - injury from a needle that has entered the source's blood vessel
            - terminal HIV related illness in the source
        - there is insufficient evidence to suggest that double gloving reduces risk of sharps injury
    - whether the source is high risk
        - any previous results for hepatitis B or C or HIV
        - factors placing the source in a high risk category:
            - intravenous drug user
            - sex industry worker
            - originally from sub-Saharan Africa
            - regularly has unprotected sex with any of the above
            - child whose mother has HIV
            - source has or is under investigation for and AIDS defining illness
        - if the source is unknown, the usual approach is to assume a low risk exposure
- investigations for healthcare workers
    - blood sample for storing for further testing
    - blood from the source patient, if known, for:
        - hepatitis B surface antigen
        - hepatitis C antibody
        - HIV antibody

                - results should be available within 8 hours, or definitely within 24 hours, to limit exposure to PEP
        - consent
            - the source patient should not be approached by the affected healthcare worker
            - sensitive discussion may be needed in terms of the process, how and with whom the results will be shared and the possible impact of positive results on patient health or life insurance policies
            - for patients who lack capacity, the BMA recommends:
                - if they are expected to regain capacity before testing is required, it should be delayed until then
                - if a relevant advanced decision to refuse treatment is not in place
                    - in England, Wales and Northern Ireland, a doctor must assess whether testing is within the best interests of the patient
                    - in Scotland the doctor must assess whether testing will benefit the patient and is reasonable in the circumstances to safeguard or promote their mental health
                - in England, Wales and Northern Ireland, where there is no potential benefit to the patient, in the absence of evidence otherwise, it is safe to assume that patients would want to 'do the right thing'; in Scotland, it is not clear that the legislation would support the same approach
                - if the patient regains capacity, they should be informed that the test has been taken and provided with enough information to decide whether to receive the results
- management for healthcare workers
    - immediate management
        - wash the site liberally with soap and running water
            - antiseptics or skin washes should not be used as their effect on local immunological defences is not known
            - puncture wounds should be encouraged to bleed freely but not sucked
            - exposed mucous membranes should be irrigated thoroughly with water
                - if the conjunctivae are involved, irrigation should be done before and after removal of contact lenses
    - HIV post exposure prophylaxis
        - if the source is known to be HIV positive or assessed as high risk, PEP should be considered
            - the healthcare worker's perception of risk should also be included in the assessment
            - nationally, risk of transmission is no more than 0.3%
        - if the source patient is known to be HIV positive and the vial load is known, this can further inform the decision
            - if the viral load is undetectable, PEP is not recommended

        - there may still be a theoretical risk of transmission, but it is very low – however, PEP should still be offered to those concerned about the risk
        - advice from the infectious diseases or microbiology team should be sought if further input is needed
    - if PEP is to be prescribed, it should be started as soon as possible, ideally within the hour
        - it is not recommended more than 72 hours post-exposure
    - emergency departments should have packs of 3-5 days of PEP (accounting for weekends and bank holidays) available to dispense immediately
        - they should contain:
            - Truvada (245mg tenofovir and 200mg emtricitabine) OD
            - Raltegravir (400mg BD)
        - written information about PEP should be provided (e.g. the information leaflet from the HIV pharmacy association)
    - considerations:
        - HIV PEP should not be taken with rifampicin
        - it should be taken at least 4 hours after taking any vitamin supplements, calcium, iron or magnesium supplements
        - patients using oral contraception should use barrier methods while taking PEP and awaiting results
        - if pregnancy cannot be excluded, a pre-treatment pregnancy test should be performed, but pregnancy should not preclude the use of PEP
            - there is no evidence to suggest decreased efficacy or toxicity
- hepatitis B prophylaxis
    - hepatitis B PEP is in the form of an accelerated course or booster dose of the vaccine, with or without hepatitis B immunoglobulin (HBIG)
        - unvaccinated exposed person
            - HBsAg positive source
                - accelerated hep B vaccination course
                - HBIG with first dose
            - source unknown
                - accelerated hep B vaccination course
            - HBsAg negative source
                - consider course of hep B vaccination
        - partially vaccinated exposed person
            - HBsAg positive source
                - one dose of hep B vaccine and finish course
            - source unknown
                - one dose of hep B vaccine and finish course
            - HBsAg negative source
                - complete course of hep B vaccine
        - fully vaccinated with primary course
            - HBsAg positive source
                - hep B vaccine booster if last dose ≥1 year ago
            - source unknown

                - consider hep B vaccine booster if last dose ≥1 year ago
            - HBsAg negative source
                - no hep B prophylaxis
        - known non-responder to hep B vaccine
            - HBsAg positive source
                - HBIG
                - hep B vaccine booster
                - second dose of HBIG at one month
            - unknown source
                - HBIG
                - consider hep B vaccine booster
                - second dose of HBIG at one month
            - HBsAg negative source
                - no HBIG
                - consider hep B vaccine booster
    - hepatitis B immunoglobulin
        - administered as an IM injection
        - doses:
            - adults and children over 10 years: 500 IU
            - children aged 5-9: 300 IU
            - newborn and children 0-4 years: 200 IU
        - provides passive immunity and can give immediate but temporary protection
        - has no effect on long term active immunity
        - prepared from donated plasma of immunised and screened human donors
            - usually available from the blood transfusion service
    - hepatitis B vaccination
        - Energix B and HBVaxPro are available in the UK
        - doses vary by age and renal function
- hepatitis C
    - there is currently no PEP for hepatitis C
    - if a source patient is found to be hepatitis C positive, the healthcare worker will be followed up by occupational health
    - 82% of HIV infected individuals are co-infected with hepatitis C
- incident reporting
    - important to prompt individual management and to ensure local practices are reviewed to minimise risk of subsequent occupational injury
    - there is a significant rate of under-reporting of needlestick injuries and exposures
    - reasons for lack of reporting among healthcare workers include:
        - they presume risk of disease transmission is low
        - there is a lack of knowledge of systems for reporting or local protocols are complicated or unclear
        - there is a lack of knowledge of the importance of reporting
        - there is a belief that reporting may reflect badly on their standards of practice

- needlestick injury in the community
    - RCEM recommends that the ED should provide initial care to these patients
    - hypodermic needles may be intentionally shared among intravenous drug users
    - discarded needles may be encountered in public spaces or the home
    - inappropriately disposed of needles and sharps may be encountered in any activity that involves handling waste
    - in most cases it will not be possible to ascertain:
        - whether the needle is contaminated and what it has been used for
        - the BBV status of the source
        - the time between the needle use and the needlestick injury
    - investigations
        - blood should be sent from the recipient for:
            - hepatitis B surface antigen
            - hepatitis C antibody
            - HIV antibody
    - if the donor also attends the ED and is willing to undergo investigation, their blood should be sent for the same investigations if BBV status is unknown
        - if blood results will be available within 72 hours and there is a reliable way of following up, it may be appropriate to delay treatment decisions until results are available
    - HIV
        - once blood has dried, HIV becomes non-viable in 2 hours
        - in general HIV PEP is not recommended following a community needlestick injury
        - where needle sharing has occurred a risk assessment considering the likelihood of the individual being HIV positive should be done; in the UK this includes:
            - men who have sex with men
            - originally from sub-Saharan Africa
            - IV drug user from a country where there is a high prevalence of HIV amongst IVDUs
                - prevalence is particularly high in Eastern Europe and Central Asia
    - hepatitis B
        - should be prescribed and administered as per advice for healthcare workers
        - hep B vaccine has been part of the routine schedule from 1st August 2017
            - anyone born before then will only have had the vaccine if they are in a high risk group and have requested it or been offered it by their GP or a sexual health service
            - the first dose of the vaccine should be given in the ED if needed and the following doses given via the GP
- discharge advice
    - pending investigation results and follow up, any patient having had a high risk exposure should be advised to:
        - use barrier contraception for any sexual contact
        - avoid sharing razors and toothbrushes
        - not donate blood until results are back

    - healthcare workers can continue to carry out activities as normal
- follow up
    - the ED clinician's role ends after initial assessment, initial investigations and provision of PEP where required
    - follow up of healthcare workers is via occupational health
    - follow up for those presenting from the community varies between trusts
        - may be via local sexual health service, infectious diseases team or GP
        - ED guidelines must state who is responsible for follow up and there must be a reliable and sufficiently fast referral pathway

IC1 influenza

- pathophysiology
    - 3 subtypes: A, B and C
        - A: more frequent, causes major outbreaks
        - B: circulates with A in yearly outbreaks, causes less serious illness
        - C: tends to cause mild or asymptomatic illness like a common cold
    - influenza A subtypes are categorised by surface antigens
        - H: haemagglutinin – facilitates entry of virus into host respiratory cells
        - N: neuraminidase – facilitates release of virions from infected host cells
    - aquatic birds and pigs are the natural reservoir
        - new types are likely to emerge where there is close co-habitation with humans
    - the virus undergoes minor mutations to one or both of the surface antigens
        - antigenic drift
        - causes seasonal epidemics with only partial immunity from previous infections
    - influenza A can have major sudden changes in the H and N antigens producing a new subtype
        - antigenic shift
        - may cause a major epidemic as little population immunity
- incidence
    - up to 15% of the population per year
    - highly infectious, ratio of infections to clinical cases of between 3:1 and 9:1
    - an epidemic may be declared when the rate of GP consultations for flu or flu-like illness is >400 per 100,000 per week
    - WHO definition of pandemic:
        - disease must be new to the population (or not have surfaced for a long time)
        - must be caused by disease-causing agents that infect humans, causing serious illness
        - the agents must spread easily and sustainably amongst humans
- risk factors
    - closed environments (e.g. schools, residential homes, prisons)
    - advanced age
    - pre-existing cardiac or respiratory illness
- transmission
    - droplet due to coughing or sneezing

    - direct nasal or eye contact with hands carrying the virus
- presentation
    - incubation period of 1-3 days
    - main features:
        - anorexia
        - malaise
        - headache (retro-orbital)
        - fever
        - myalgia
        - non-productive cough and sore throat
    - nasal discharge/obstruction and sneezing can occur but are not normally prominent features
    - older patients may not have fever
    - GI symptoms are rare
    - most symptoms last 3-5 days, with cough, tiredness and malaise lasting up to 1-2 weeks
    - infectivity continues for 5 days from onset
        - children can remain infectious for 2 weeks
        - immunocompromised patients can be infectious for weeks
    - atypical symptoms in children
        - H1N1
            - haematemesis
            - photophobia
            - chest pain
            - epistaxis
            - croup
            - apnoea
            - rigors
            - in young children and babies:
                - apnoea
                - reduced tone
                - poor feeding
                - sudden severe collapse (ALTE)
        - all influenzas
            - lethargy
            - poor feeding
            - apnoea
            - fever
            - pneumonia
            - otitis media
            - drowsiness (50% of under 4s)
- investigation
    - nasopharyngeal viral swab
- management
    - self-care in otherwise healthy people
    - pharmacological

        - neuraminidase inhibitors (e.g. oseltamivir, zanamivir) can reduce symptoms by around 1 day and need to be started within hours of symptom onset
        - may be considered for at risk patients during local outbreaks if it can be started within 48 hours of symptom onset or of exposure (as prophylaxis)
- complications
    - respiratory
        - acute bronchitis
        - secondary bacterial pneumonia
        - primary viral pneumonia
        - exacerbations of asthma and COPD
        - empyema
        - pulmonary aspergillosis
        - sinusitis
    - non-respiratory complications
        - febrile convulsions
        - otitis media
        - toxic shock syndrome
        - myositis and myoglobinaemia
        - heart failure
        - myocarditis
        - Reye's syndrome
        - Guillain-Barré syndrome
        - transverse myelitis
        - encephalitis

IC2 infection in immunocompromised patients

- background
    - immunosuppression is increasingly common
        - e.g. secondary to chemotherapy, stem cell transplant, solid organ transplant, therapies for autoimmune diseases
    - patients are susceptible to common pathogens and opportunistic pathogens
- presentation
    - inflammatory responses are impaired
        - may lack fever
        - may lack localising signs of infection
        - may lack typical radiographic changes
        - invasive diagnostic tests may be required
        - infection may be difficult to distinguish from rejection and graft-vs-host disease
- management
    - early use of appropriate antibiotics is critical
    - drug interactions and side effects are common
    - consider parasitic diseases in patients from endemic areas
    - many viral infections lack effective treatments
    - resistance to antimicrobials is common
- Pneumocystis pneumonia
    - pathophysiology

        - pneumonia caused by yeast like fungus Pneumocystis jirovecii
        - most common in the immunosuppressed
        - AIDS-defining illness in HIV patients
        - previously thought to be P. carinii and has been reclassified
        - Pneumocystis spp cannot be cultured
    - clinical features
        - subtle onset of progressive dyspnoea, non-productive cough, low grade fever
        - abrupt onset of respiratory insufficiency, may correlate with increased or tapered dosage of immunosuppressant drugs in non-AIDS patients
        - sudden onset chest pain and dyspnoea suggests pneumothorax
    - investigations
        - induced sputum or BAL for PCR
        - test for HIV and CMV (co-infection is common)
        - CXR
            - bilateral symmetric interstitial or granular opacities, pneumatocoeles, pneumothorax (may be bilateral)
        - HRCT chest
            - ground glass opacities
    - management
        - co-trimoxazole for 21 days (IV or oral)
        - steroids for patients with HIV and significant hypoxaemia
        - long term chest drains may be required for pneumothorax
    - prognosis
        - 10-20% mortality for initial PCP infection in AIDS patients, higher if requiring mechanical ventilation
        - 30-60% mortality for initial infection in patients without AIDS
- post transplant infection
    - early (<1 month)
        - related to:
            - technical factors
            - nosocomial infections
            - donor-derived infections
            - recipient colonisers
    - types
        - active infections
            - transmitted with the allograft; commonly seed the allograft, especially at anastomoses
            - e.g. MRSA, VRE, candida
            - active bacterial and fungal infections in the recipient must be eradicated before transplantation
        - pulmonary infections
            - aspiration and postsurgical
        - infections in devitalised tissues or undrained fluid collections – high risk for microbial seeding
        - C. difficile colitis
    - medium (1-6 months)

        - residual infections from the first month
        - immunomodulating viruses
            - CMV, EBV, HSV, human herpes virus 6, HBV, HCV, BK virus
            - often activation of latent infection
        - opportunistic infections
            - Pneumocystis carinii, Aspergillus, L.moncytogenes
            - parasites in endemic regions (e.g. Toxoplasma, Strongyloides, Leishmania)
    - longterm (>6 months)
        - community acquired infection in those with good allograft function
        - chronic and/or progressive viral infections
        - recurrent or chronic infection with decreased allograft function and needing high dose immunosuppression
            - opportunistic pathogens
            - may need lifelong prophylaxis

IC3 infestations

- scabies
    - pathophysiology
        - caused by the parasitic mite Sarcoptes scabiei
        - large global public health problem leading to the formation of the International Alliance for the Control of Scabies (IACS)
        - female scabies mite is 0.4mm long, male is half this size
        - after mating on the skin surface, the male dies and the female tunnels into the epidermis and lays eggs along the burrow
        - development from egg to adult takes 10-15 days, and the adults return to the skin surface to multiply
        - skin contact of 10-15 minutes usually required for mites to move between hosts; females do not jump or fly
            - crusted scabies can also be transmitted via bedding, clothes, furniture, towels
        - there is an asymptomatic period, with pruritus developing as an allergic reaction at 4-6 weeks
            - infection can be transmitted during the asymptomatic phase
    - risk factors
        - poverty
        - overcrowding
        - malnutrition
        - homelessness
        - poor hygiene
        - institutions
            - e.g. residential homes, refugee camps
        - dementia
        - sexual contact
        - children
        - immunosuppression
    - presentation

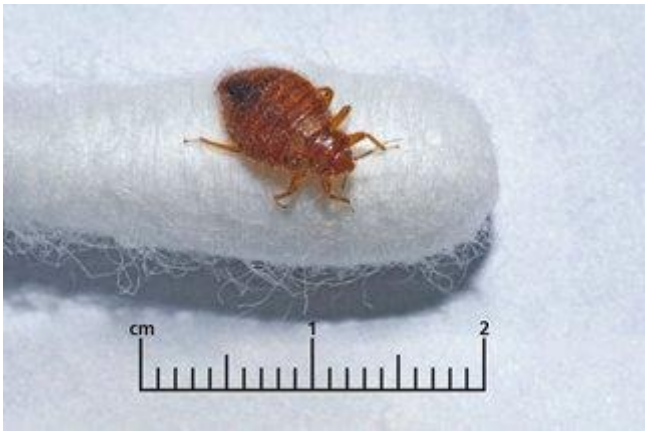
https://www.nhs.uk/conditions/scabies/

- signs and symptoms at 3-4 weeks
    - can occur within 1-3 days with reinfestation due to prior sensitisation
- most common symptom is widespread itching
    - worse at night
    - worse when warm
    - history of family members with itch is suggestive
    - can have secondary bacterial infection due to scratching
- skin changes vary
    - may be papules, vesicles, pustules, nodules

        - widespread symmetrical, itchy popular eruption most commonly seen around axillae, peri-areolar region of breasts in women, abdomen, buttocks, thighs
    - burrows may be visible
        - fine wavy greyish, dark or silvery lines
        - most often in interdigital web spaces of hands, flexor surfaces of wrists and elbows, axillae, ankles, feet, buttock areas, male genitalia, peri-areolar region
    - nodules may develop
    - thick skin (e.g. soles of feet) is relatively resistant
    - papules are small and erythematous and can change into vesicles and bullae
- crusted scabies
    - hyper-infestation with thousands of mites, presenting in exfoliating scales due to host's inefficient immune response
    - risk in immunosuppressed, elderly, those with decreased peripheral sensation
    - hyperkeratotic skin disease resembling psoriasis
    - may have generalised lymphadenopathy and eosinophilia
    - often has secondary infection
    - very contagious and difficult to eradicate
- diagnosis
    - mainly clinical
    - ink burrow test may be helpful – ink is rubbed over a burrow then wiped off with an alcohol rub, and tracks into the burrow
- management
    - all members of the household, close contacts and sexual contacts should be treated at the same time as the index patient – treatment should be applied on the same day
    - primary treatment is topical parasiticidal preparation applied head to toe and left on overnight
        - repeated one week later
        - must include scalp, neck and ears, between fingers and toes and under the nails
        - should be applied after a hot bath
        - needs to be reapplied after handwashing
        - first line is permethrin 5%, second line is malathion 0.5%
    - clothes, towels and bedlinen should be machine washed at at least 50 degrees
        - things that cannot be washed should be sealed in a bag for at least 72 hours
    - outbreaks should be referred to public health
    - itch
        - antihistamines do not help, but sedating antihistamine at night may help with sleep
        - crotamiton cream or lotion can help
        - emollients, moisturisers or low dose steroid creams kept in fridge
    - full STI screening should be offered

- complications
  - flaring or reactivation of eczema or psoriasis
  - secondary bacterial infection (can become systemic)
  - psychological harm due to stigma
- prognosis
  - persists indefinitely until treated
  - re-infestation possible
  - itch can continue for three weeks after treatment
    - if persisting for 6 weeks, consider treatment failure
  - crusted scabies may be very difficult to eliminate in patients with HIV

- bedbugs
  - pathophysiology
    - increase in past decade, likely due to increased international travel and changes in pest control practice
    - two main species: Cimex lectularius and Cimex hemipterus
    - brownish, wingless, flattened insects, 2-5mm in length
      - can survive for a year without eating
      - can crawl rapidly, cannot jump or fly
      - inactive during the day, hiding in dark places
      - live in clean or dirty environments
  - presentation



https://www.nhs.uk/conditions/bedbugs/

    - initial bite usually not felt as they bite at night and inject anaesthetic

        - can have 3 or 4 bites forming a line or curve on parts of the body exposed when sleeping
        - isolated pruritus, papules or nodules can occur
        - bullous eruptions or, rarely, systemic reactions such as urticaria or anaphylaxis
    - management
        - eradication is difficult due to hiding and long survival times
            - washing at 60 degrees or dry cleaning
            - tumble drying on hot for at least half an hour
            - freezing for at least one to two weeks
            - disposal of highly infected items
            - encasing mattresses in a sealed covering may be effective
            - guidance from professionals regarding insecticides
                - can be resistant
                - carbon dioxide fumigation for at least 24 hours can be helpful
        - symptom management
            - topical steroids for up to seven days
            - antihistamines if there is pruritus
            - topical or systemic antibiotics for secondary infections
    - complications
        - secondary infection
        - no report of infectious disease transmission, although this is suspected
        - sleep deprivation
        - psychological sequelae including nightmares, hypervigilance, anxiety
- head lice
    - epidemiology
        - Pediculus humanus capitis
        - common worldwide, endemic in the UK
            - most common in children aged 4-11
        - no evidence of preference for clean or dirty hair
        - louse eggs attach to hair shafts and take 7-10 days to hatch
        - immature lice (nymphs) mature into adults at 7-10 days
        - adult lice are around the size of a sesame seed
            - they cannot jump or fly
        - lice feed on blood several times a day
        - transmission usually requires head to head contact
    - risk factors
        - age under 12
        - female gender
        - families with four or more children
        - lower socio-economic status
        - long hair
    - presentation

https://www.nhs.uk/conditions/head-lice-and-nits/

        - often asymptomatic, may present with itch
            - itch can develop after several days/months and persist for weeks after eradication
        - nits (eggs) are not sufficient for diagnosis as dead and live eggs look the same – live lice must be found with detective combing
            - a fine toothed comb (wider gaps than a nit eradication comb) is used
            - hair conditioner immobilises the lice
    - management
        - treat only if a live louse is found
        - treat all affected household members simultaneously
        - treatment success should be confirmed
        - clothing/bedding do not need to be washed
        - children can still attend school
        - treatment options include:
            - mechanical methods (e.g. wet combing)
                - four sessions over two weeks
                - 50-60% cure rate, less effective than insecticides
            - physical insecticides – coat the lice and block the oxygen supply
                - e.g. dimeticone 4% lotion
                - applied twice 7 days apart
            - chemical or traditional insecticides – have a neurotoxic action and poison the lice
                - e.g. malathion 0.5% aqueous liquid
                - applied twice 7 days apart
                - resistance has developed to previous insecticides such as permethrin
    - complications
        - pruritic rash on back of neck and behind ears
            - hypersensitivity to louse faeces
        - excoriation, skin infection and impetigo
        - loss of sleep due to itching
        - anxiety and distress
- pubic lice

- pathophysiology
  - Pthirus pubis
    - crab-shaped, grey-brown, 2mm in length
    - female lays eggs near the hair shaft which hatch after 6-10 days
    - common among young adults
    - transmitted by close body contact (sexual or close family contact)
      - may be an indication of sexual abuse in children but more commonly contracted innocently
- presentation
  - discovery of adult lice or eggs
  - live on coarse hair, especially pubic/perianal
    - also on eyelashes, abdomen, back, axillae, head
    - fine toothed comb or dermatoscopy can be used
  - itchy red papules are the most common presentation
    - itching occurs 1-3 weeks after infestation but can occur immediately on reinfestation
    - tends to be worse at night
  - blue macules may be visible at feeding sites
  - tiny dark brown specks of louse excreta may be seen on underwear or skin
- management
  - GUM clinic referral if sexually acquired
    - for treatment, STI screening and contact tracing
  - topical insecticide, two applications 7 days apart
  - shaving does not prevent reinfestation
- complications
  - excoriation and skin infection due to scratching
  - blepharitis, conjunctivitis epithelial keratitis when eyelashes involved

- body lice
  - pathophysiology
    - Pediculus humanus
    - usually caught by contact with infested person
    - seen most often in cold climates, poor sanitation, overcrowding
    - occur mainly when clothes are infrequently washed
    - nits are laid in host clothing
    - may co-exist with scabies
  - presentation
    - often asymptomatic, patients often present when lice or eggs are found
    - pruritus and excoriation
    - can be found in any body region, tend to avoid the scalp
  - management
    - improved hygiene, hot water laundering of clothes and linen
    - contacts must be treated at the same time
    - if not sufficient, may require permethrin or malathion
    - treating clothing with permethrin may prevent infestation
  - complications
    - vector for trench fever (Bartonella quintana), epidemic typhus and relapsing fever

- prognosis
    - treatments effective at killing nymphs and mature lice, less so eggs
    - cure effective in 90% of cases

IC4 Kawasaki disease

- pathophysiology
    - also known as Mucocutaneous Lymph Node Syndrome
    - idiopathic self-limiting vasculitis
    - commonest cause of acquired childhood heart disease in developed world
    - incidence in Europe 5-10 per 100,000 children under 5
    - more common in boys, under 5s and Asians
        - can occur at any age including adults
    - diagnosis frequently missed as it presents like a viral infection
    - aetiology unknown, likely mixture of infectious and genetic
        - assumption is that an infection triggers an abnormal immune response
    - affects small and medium sized arteries in multiple organs and tissues
- diagnosis (children under 1 may have fewer features)

https://www.heart.org/en/health-topics/kawasaki-disease/kawasaki-disease-signs-symptoms--diagnosis

https://www.rch.org.au/uploadedImages/Main/Content/clinicalguide/guideline_index/5.png

    - fever of abrupt onset (usually >39°, present for 5 days – experienced clinicians may diagnose earlier)

- plus four of:
- bilateral, nonexudative bulbar conjunctival injection
    - bilateral scleral injection with limbal sparing (clear zone around iris)
- oropharyngeal mucous membrane changes
    - pharyngeal erythema
    - red/cracked lips
    - strawberry tongue
- cervical lymphadenopathy
    - at least one node >1.5cm in diameter
- peripheral extremity changes
    - acute phase: diffuse erythema and swelling of hands and feet
    - convalescent phase: periungual desquamation (weeks 2-3)
- polymorphous generalised rash
    - nonvesicular, nonbullous
    - no specific pathognomic rash
  - signs may occur sequentially rather than concurrently – good history required with high index of suspicion
- phases of illness
  - acute
    - 1-2 weeks
    - highly febrile
    - very irritable
    - toxic-appearing
    - oral changes
    - oedema and erythema of hands and feet
    - rash especially common in perineal area
  - subacute
    - 2-8 weeks
    - gradual improvement
    - fever settles
    - desquamation of perineum, palms, soles
    - arthritis, arthralgia
    - thrombocytosis
    - coronary artery aneurysms
    - myocardial infarction
  - convalescent
    - months to years
    - resolution of remaining symptoms
    - laboratory values return to normal
    - aneurysms may resolve or persist
    - Beau's lines
    - cardiac dysfunction and myocardial infarction may still occur
- investigations
  - rule out other causes
  - echocardiography
    - often serial as illness evolves
  - non-specific findings include:

        - normochromic anaemia and leucocytosis
        - thrombocytosis in second week
        - LFT changes and hypoalbuminaemia
        - raised CRP/ESR
        - sterile pyuria
- complications
    - cardiac
        - carditis during febrile phase
            - myocarditis with ST-T changes (25%)
            - pericardial effusion (20-40%)
            - valvular dysfunction (1-2%)
            - cardiac failure (5%)
        - coronary vessel abnormalities
            - 20% of cases if untreated, <5% if treated
            - peaks at 2-4 weeks
            - aneurysm formation may lead to fatalities from thrombosis, rupture or ischaemia-related dysrhythmia
    - common associations (vasculitis may affect any organ)
        - arthritis
        - keratitis/uveitis
        - diarrhoea, vomiting, gallbladder disease
        - coryza and cough
- management
    - IV immunoglobulin
        - 2g/kg IV over 10 hours
        - ideally within 10 days of onset
        - second dose may be given if fevers persist
    - aspirin
        - 3-5mg/kg PO daily for 6-8 weeks
        - some recommend initial higher doses
        - evidence on dose and whether aspirin is useful is lacking
    - corticosteroids
        - sometimes used in resistant cases but little evidence
- prognosis
    - depends on degree of cardiac involvement
    - most coronary artery aneurysms (50-70%) regress after 1-2 years
        - giant aneurysms never resolve completely and have a worse prognosis

IC5 notifiable diseases

- registered medical practitioners have a statutory duty of notification:
    - complete a notification form immediately on diagnosis of a suspected notifiable disease
        - without waiting for laboratory confirmation
    - send the form to the proper office within 3 days or notify verbally within 24 hours if urgent
- there is widespread under-reporting and lack of compliance in the UK and abroad
- details required for notification:

    - patient's name, date of birth, sex, home address with postcode
    - patient's NHS number
    - ethnicity
    - occupation and/or place of work or education
    - current residence if different from home address
    - contact telephone number
    - contact details of parent if patient is a child
    - disease or infection or nature of poisoning/contamination
    - date of onset of symptoms and date of diagnosis
    - any relevant overseas travel history
    - if in hospital, hospital address, day admitted, whether disease was contracted in hospital
- list of notifiable diseases in England:
    - anthrax
    - botulism
    - brucellosis
    - cholera
    - Covid-19
    - diphtheria
    - encephalitis and meningitis
    - enteric fever
        - typhoid or paratyphoid
    - food poisoning
    - haemolytic uraemic syndrome
    - hepatitis (infectious)
    - infectious bloody diarrhoea
    - invasive group A streptococcal disease
    - Legionnaire's disease
    - leprosy
    - malaria
    - measles
    - meningococcal septicaemia
    - mumps
    - plague
    - poliomyelitis
    - rabies
    - rubella
    - scarlet fever
    - severe acute respiratory syndrome
    - smallpox
    - tetanus
    - tuberculosis
    - viral haemorrhagic fever
    - whooping cough
    - yellow fever

IC6 pyrexia of unknown origin – different age groups

- unlikely to be an ED diagnosis

- definition:
    - temperature >38.3° on several occasions
    - more than 3 weeks of illness
    - no diagnosis after a week of inpatient investigation
- additional categories:
    - nosocomial PUO in hospital patients with fever of 38.3° on several occasions caused by a process not present or incubating on admission and cultures negative and diagnosis unknown after 3 days of investigation
        - common causes include medication, postoperative complications, septic thrombophlebitis, pulmonary emboli, MI, stroke, blood transfusion reactions and C. diff
    - neutropaenic PUO – fever with <1 $x10^9$ neutrophils, initial negative cultures and uncertain diagnosis after 3 days
    - HIV-associated PUO in patients with HIV and fever for four weeks as outpatients or three days as inpatients with uncertain diagnosis after 3 days of investigation
- common causes
    - most are unusual presentations of common disease
        - e.g. tuberculosis, endocarditis, gallbladder disease, HIV
    - in adults, infections and cancer (25-40% of cases each) account for most PUOs
        - autoimmune disorders account for 10-20% of cases
    - in children, infectious disease (37.6%) is the main cause, followed by malignancy (17.2%), miscellaneous disease (16-1%) and collagen vascular disease (14%)
- history
    - all symptoms from all major systems plus general complaints
    - previous illnesses including surgery and psychiatric problems
    - nutrition including dairy products and their source
    - drug history including OTC and illicit
    - immunisation status
    - family history
    - occupational history including exposure to chemicals/animals
    - travel and recreational habits (including exposure to ticks and other vectors)
    - sexual history
- ED investigations:
    - FBC, U&E, LFT, clotting, ESR, CRP
    - blood cultures
        - at least 2 sets from different sites
    - sputum culture and microscopy
    - CXR
    - ECG
    - urine dip, microscopy and culture
    - stool culture
    - other investigations may include:
        - thick and thin films if travel to malarial region in past year
        - glandular fever screen
        - HIV serology (with appropriate counselling/consent)
        - syphilis serology
        - echo if endocarditis is a possibility

        - further imaging if source remains occult (e.g. CT TAP)
        - autoantibodies
        - hepatitis screen
- ED management
    - empirical antibiotics if infective source suspected
        - after samples and cultures taken
    - antipyretics
        - for symptom relief
        - to reduce metabolic consequences such as dehydration, increased oxygen consumption and increased metabolic rate
    - increased fluid intake
        - may require IV fluid
    - removal of excess clothing and bed linen
- examples of causes
    - bacterial
        - abscesses
        - TB
        - UTIs
        - endocarditis
            - HACEK group responsible for 5-10% (Haemophilus, Actinobacillus actinomycetemcomitans, Cardiobacterium hominis, Eikenella corrodens, Kingella)
        - hepatobiliary infections
        - osteomyelitis
        - brucellosis
        - Borrelia recurrentis (tick borne)
        - other spirochaetes (e.g. Lyme disease, syphilis)
    - viral
        - herpes viruses (e.g. CMV, EBV)
        - HIV
            - usually due to infections or lymphoma
    - fungi
    - parasites
        - toxoplasmosis
        - malaria
        - trypanosoma, leishmania and amoeba species
    - neoplasm
        - Hodgkin's and non-Hodgkin's lymphoma
        - leukaemia
        - renal cell carcinoma or other solid tumours
    - drugs
        - beta lactam antibiotics, isoniazid
    - collagen vascular and autoimmune
        - systemic onset juvenile idiopathic arthritis
        - polyarteritis nodosa
        - rheumatoid arthritis
        - mixed connective tissue diseases
    - granulomatous disease

        - sarcoidosis
        - Crohn's
    - vasculitides
    - pulmonary emboli and thrombophlebitis
    - hyperthyroidism
- PUO in children
    - Kawasaki disease needs to be considered
    - fever for longer than 5 days is an amber flag in the NICE traffic light system and admission should be considered

IC7 malaria

- anyone who presents with a fever within a year of visiting an endemic area should be considered to potentially have malaria
    - endemic areas are in Africa, India, New Guinea, South East Asia and tropical climate areas
- types
    - five species infect humans
        - Plasmodium vivax, malariae, ovale, falciparum, knowlesi
    - falciparum
        - most pathogenic and resistant to standard antimalarials
        - can be rapidly fatal
        - lacks a liver phase (hypnozooites)
        - typically develops within 6 weeks of return from endemic area (presentation can be delayed up to 6 months if on prophylaxis)
    - others
        - vivax, ovale and malariae can cause significant distress but tend to have a benign prognosis
        - vivax and ovale have hypnozooites so need treatment to prevent relapse from reactivation of these
        - knowlesi is a simian disease and considered a zoonosis
            - potentially lethal, should be treated like falciparum
- risk factors for severe malaria
    - pregnancy
    - splenectomy
    - extremes of age
- history
    - travel history, usually within past six months
        - including countries visited, stopovers, date of return
        - prophylaxis taken, adherence and date of stopping
- symptoms
    - fever, may recur and be cyclical
    - chills
    - rigors
    - headache
    - cough
    - myalgia
    - GI upset

- signs
    - fever
    - splenomegaly
    - hepatomegaly
    - jaundice
    - P malariae can cause nephrotic syndrome
- severe malaria
    - CNS (cerebral malaria): altered consciousness, meningism, seizures, focal deficits
    - GI: vomiting, jaundice
    - resp: respiratory distress +/- ARDS
        renal: AKI
    - CVS: shock
    - haem: severe haemolytic anaemia with jaundice, coagulopathies, spontaneous haemorrhage
    - metabolic: acidosis and hypoglycaemia
- investigations
    - general
        - glucose
        - FBC, U&Es, LFTs, coagulation, group and save
        - haemolysis screen
        - septic screen to exclude other causes
        - consider CT head +/- LP
        - EEG if suspected seizures
        - CXR
    - specific
        - thick and thin blood films
            - EDTA tubes
            - performed urgently – need to alert lab out of hours
            - thick film to detect parasites
            - thin film to determine species and quantify
            - multiple samples should be sent, usually three samples 12 hours apart
                - one negative film does not rule out
            - may be negative if patient on antimalarials or recent antibiotics
        - immune-chromatograph testing
        - PCR
- management
    - general
        - early ID input
        - don't treat with the same agent the patient was on for prophylaxis
        - check resistance patterns of endemic region
        - urgent treatment required if:
            - any degree of altered consciousness, jaundice, oliguria, severe anaemia or hypoglycaemia
            - parasite count . 100,000/mm^3 or >2% red blood cells parasitised
            - vomiting or acidotic
    - specific

        - severe falciparum malaria
            - IV artesunate + lumefantrine
            - IV quinine
                - watch for hypoglycaemia, arrhythmia, cinchonism
        - uncomplicated falciparum
            - PO quinine and doxycycline for 7 days
            - atovaquone-proguanil (Malarone)
            - artemether with lumefantrine
        - vivax, malariae and ovale
            - chloroquine +primaquine to clear liver disease
    - most patients should be admitted for ID review
    - malaria is a notifiable disease

IC8 HIV infection

- pathophysiology
    - lentivirus from the subfamily of retroviruses
    - first identified in 1983
    - causes acquired immune deficiency syndrome (AIDS)
    - binds to CD4 receptors on helper T cells
        - CD4 cells migrate to lymphoid tissue
        - virus replicates and infects new CD4 positive cells
        - depletion or impaired function of CD4 cells predisposes to immune dysfunction
- CDC stages of HIV
    - Stage 1: acute primary infection (seroconversion illness)
        - between one and six weeks after infection (20-60% of people)
        - common symptoms:
            - glandular fever type illness
                - fever, malaise, myalgia, pharyngitis, headaches, diarrhoea, neuralgia or neuropathy, lymphadenopathy, maculopapular rash
            - may be asymptomatic
        - consider HIV seroconversion if unusual signs
            - e.g. oral candidiasis, recurrent shingles, leukopaenia, CNS signs
            - early recognition is more important now there are good treatments available
        - viral p24 antigen and HIV RNA levels are elevated (antibodies are negative)
    - Stage 2: asymptomatic stage
        - virus levels are low after seroconversion
        - replication continues slowly
        - CD4 and CD8 lymphocyte levels are normal
        - may persist for years
    - Stage 3: symptomatic HIV infection
        - nonspecific constitutional symptoms
            - fever, night sweats, diarrhoea, weight loss
        - may be minor opportunistic infections

                - e.g. oral candida, oral hairy leukoplakia, herpes zoster, recurrent herpes simplex, seborrhoeic dermatitis, tinea infections
            - referred to as AIDS-related complex and is a precursor to AIDS
        - AIDS
            - severe immunodeficiency
            - serious opportunistic infections
            - evidence of life-threatening infections and unusual tumours
- manifestations by system
    - resp: PCP, aspergillosis, herpetic infections, candidiasis, CMV, TB, normal bacterial pneumonias, nocardia, lymphoma, Kaposi sarcoma
    - CVS: endocarditis, CCF, arrhythmias secondary to brainstem involvement, myocarditis
    - GI: diarrhoea, vomiting, dysphagia
    - CNS: toxoplasmosis, cryptococcus, candida, mycobacterium, treponema, aspergillus, encephalitis, aseptic meningitis, HSV, multifocal leukoencephalopathy, lymphoma, polyneuropathy, HIV dementia
    - haem: anaemia, thrombocythaemia, leucopaenia
    - endo: CMV adrenalitis, adrenal suppression secondary to steroid use
    - obstetrics: without intervention there is transmission rate of 30% during pregnancy and 20% through breast milk; with antiretroviral and c-section risk can be reduced to 2%
    - pain: numerous aetiologies including HSV, arthralgia, peripheral neuropathy, drug-related pain
- diagnosis
    - based on antibody detection in serum
    - rapid tests in some GUM clinics can produce results in 20 minutes
    - combination test for p24 antigen and HIV antibody
        - very accurate
        - provided by hospitals
        - takes around 4 weeks to get results
    - counselling required before and after testing
- testing recommendations
    - people in groups at increased risk of HIV exposure
        - e.g. men who have sex with men and their female partners, black Africans, people who inject drugs, sex workers, prisoners, transwomen, people from countries with high prevalence and their partners
    - people attending services whose users have an increased risk
        - e.g. GUM clinics, TB, hepatitis and lymphoma clinics, antenatal or termination clinics, substance abuse services
    - all people with symptoms or signs suggestive of HIV/AIDS
    - people accessing healthcare in areas with high or very high HIV seroprevalence
    - sexual partners of patients diagnosed with HIV
- AIDS-defining conditions in adults
    - candidiasis
        - bronchi, trachea, lungs or oesophagus
    - invasive cervical carcinoma
    - coccidiodomycosis
    - cryptococcosis, extrapulmonary

    - cryptosporidiosis, chronic intestinal
    - CMV other than liver, spleen or nodes; CMV retinitis with loss of vision
    - encephalopathy
    - herpes simplex, chronic or bronchitis, pneumonitis, oesophagitis
    - histoplasmosis
    - isosporiasis, chronic intestinal
    - Kaposi's sarcoma
    - lymphoma, including Burkitt's and brain
    - mycobacterium including TB
    - Pneumocystis jirovecii pneumonia
    - recurrent pneumonia
    - progressive multifocal leukoencephalopathy
    - salmonella septicaemia, recurrent
    - toxoplasmosis of brain
    - wasting syndrome due to HIV
- management of HIV
    - usually uses 3 agents
        - nucleoside reverse transcriptase inhibitors (NRTI – zidovudine)
        - nucleotide reverse transcriptase inhibitors (RTI – tenofovir)
        - non-nucleoside reverse transcriptase inhibitors (NNRTI – nevirapine)
        - protease inhibitors (PI – saquinavir)
        - others – enfuvirtide
    - aim is to achieve an undetectable viral load

MAXILLOFACIAL/DENTAL

MaP1 dental pain

- tooth descriptors
    - maxillary teeth
        - upper row
    - mandibular teeth
        - lower row
    - facial
        - portion of tooth seen when the mouth is open/smiles
        - applicable to all teeth
        - labial
            - facial surface of the incisors and canines
        - buccal
            - facial surface of the molars and premolars
    - oral
        - portion of tooth that faces the tongue or palate
        - applies to all teeth
        - lingual
            - toward the tongue; the oral surface of the mandibular teeth
        - palatal
            - toward the palate; the oral surface of the maxillary teeth

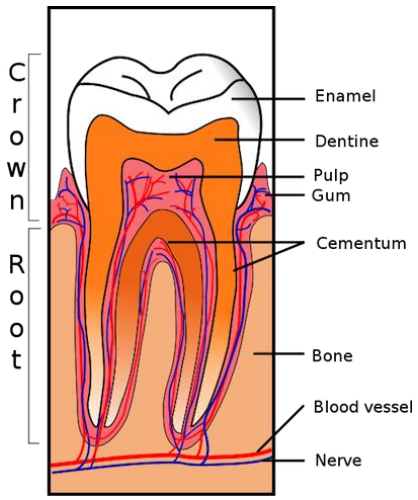
    - apical
        - toward the tip of the root of the tooth
    - coronal
        - toward the crown or the biting surface of the tooth
        - occlusal
            - biting or chewing surface of the premolars or molars
        - incisal
            - biting or chewing surface of the incisors and canines
    - approximal/interproximal
        - contacting surfaces between two adjacent teeth
        - mesial
            - interproximal surface facing anteriorly or closest to the midline
        - distal
            - interproximal surface facing posteriorly or away from the midline
- dentoalveolar injuries
    - dental fracture
    - dental subluxation
    - dental avulsion
    - bleeding dental socket
- odontogenic infections
    - acute alveolar osteitis (dry socket)
    - acute necrotising ulcerative gingivitis (trench mouth)
    - dental abscess
        - periapical abscess
        - periodontal abscess
    - Ludwig's angina
    - pulpitis (dental caries)
    - pericoronitis
    - peritonsillar abscess
    - retropharyngeal abscess
    - Vincent's angina (tonsillitis and pharyngitis)
- other
    - scurvy
    - gingival hyperplasia
        - phenytoin
        - cyclosporine
        - nifedipine
        - amlodipine
        - leukaemia

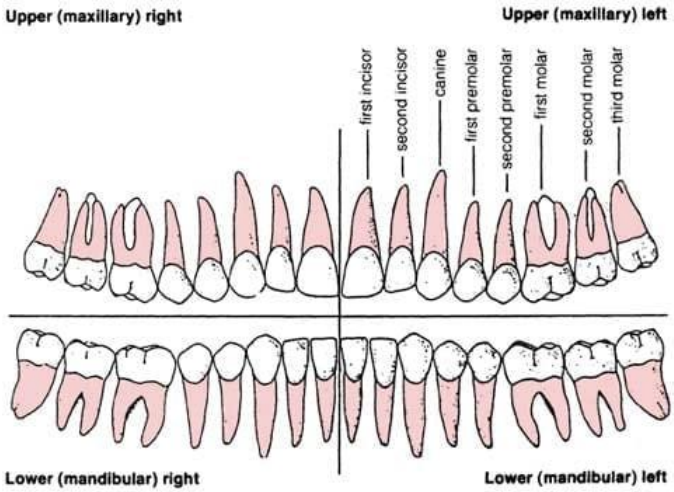
MaP2 facial swelling

- causes
    - buccal space infections
    - dental problems
    - canine space infection
    - facial cellulitis
    - herpes zoster

- masticator space infections
- maxillofacial trauma
- neoplasm
- parapharyngeal space infection
- salivary gland pathology
    - parotitis
        - bilateral parotitis
        - suppurative parotitis
        - viral parotitis (mumps)
    - ranula
    - sialoadenitis
    - sialolithiasis
- superior vena cava syndrome
- angioedema
- surgical emphysema

MaP3 avulsed or fractured teeth





https://litfl.com/tooth-avulsion/

- history
    - mechanism of injury and associated injuries

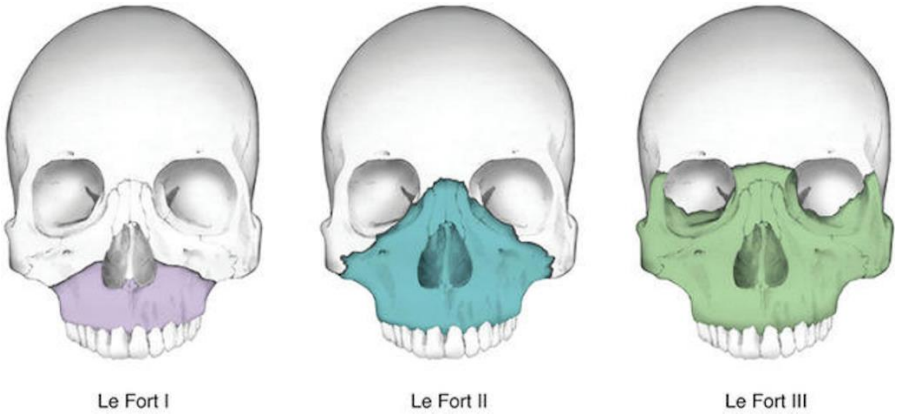
    - previous dental history
        - including injuries, crowns or prostheses
    - time since injury
    - location of permanent tooth fragments
- examination
    - symmetry in the mouth
    - bite – check for subjective or objective malocclusion
    - TMJs
    - numbness
    - intra- or extra-oral bruising
    - bony steps in maxilla or mandible
    - gingival or oral mucosal injury (lift lips to check)
    - type of tooth and whether first or permanent
    - type of dental injury
    - all lost teeth and fragments should be accounted for – check chest and soft tissues of mouth
- primary vs permanent teeth
    - primary: small, very white, bulbous crowns, often worn, flat edges
    - permanent: larger, creamier in colour, jagged edges on newly erupted teeth
        - permanent incisors usually erupt between the ages of 6 and 8 years
- investigations
    - OPG if considering fractured mandible or TMJ injury
    - CXR if aspirated tooth suspected or patient unconscious
- management of avulsed tooth
    - reinsertion within a few hours of injury
        - do not reimplant primary teeth
            - can interfere with eruption of permanent tooth
    - tooth should be manipulated by the crown only, avoid touching the root
    - tooth should be cleaned with saline and placed in storage medium
        - preferred medium is milk, preferably with ice pack around milk for transport
        - saline or saliva can be used if milk not available
        - avoid keeping tooth in buccal pouch – risk of swallowing or inhaling
    - socket should be packed with sterile gauze
    - irrigate socket with saline – local anaesthetic can also be used before reinsertion
    - tooth needs splinting after reinsertion
        - Blu-tack or aluminium foil can be moulded around tooth as a temporary measure
    - patients should not eat or drink and should be referred urgently to max-facs/dental surgeon
- dental fractures
    - Ellis classification system
        - class I
            - anatomy
                - break in superficial enamel only
            - presentation
                - chalky white fracture line
                - painless to temperature, air, percussion

        - management
            - file sharp edges with Emery board
            - routine follow up with dentist for cosmetic repair
            - soft foods
    - class II
        - anatomy
            - break through enamel with visible dentine
        - presentation
            - ivory or pale yellow fracture line
            - may be sensitive to heat, cold or air
            - not tender
        - management
            - goal is to prevent bacterial pulp contamination through exposed dentine
            - exposed dentine should be covered with calcium hydroxide or cyanoacrylate tissue adhesive then cover with dental cement or dental foil
            - analgesia, antibiotics and 48 hour dental follow up
    - class III
        - anatomy
            - break through enamel and dentine with visible pulp
        - presentation
            - pulp has pinkish, red, fleshy hue
            - frank bleeding or pink blush after wiping tooth surface indicates pulp violation
            - may be exquisitely painful or desensitised (with associated neurovascular disruption)
        - management
            - dental emergency, needs review in ED
            - if patient cannot be seen immediately, cover with calcium hydroxide and dental cement or foil
            - analgesia and antibiotics
            - for brisk bleeding, patient can bite on gauze soaked with local anaesthetic with adrenaline
- subluxation and luxation
    - subluxation: tooth is not displaced from socket but is mobile
    - luxation: tooth is partially displaced from the socket
    - management:
        - reposition tooth gently and splint
        - needs 24 hour dental follow up and a soft diet
- intrusion
    - tooth is displaced apically
    - management:
        - deep (>3mm) intrusion and/or underlying alveolar bone fracture is a dental emergency
        - requires urgent repositioning and stabilising
        - assess for concomitant injuries

        - discharge on soft diet with 24 hour dental follow up

MaP4 facial bone injury

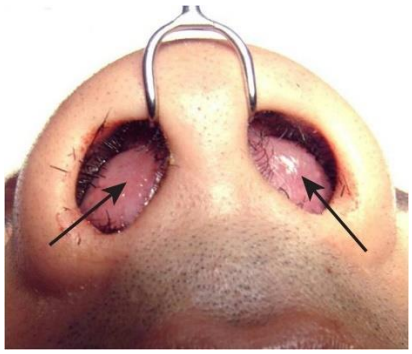
- injury types
    - mandibular fracture
    - mid facial fracture (Le Fort)
    - TMJ disruption
    - zygomatic, orbital and nasal fractures
    - soft tissue injuries
    - haemorrhage
    - associated injuries
        - base of skull fracture
        - traumatic brain injury
        - c-spine fracture
        - carotid injury
- mandibular fracture
    - fractures at the vulnerable points
        - ramus, body at level of first and second molar
        - bilateral fracture can precipitate airway obstruction from posterior displacement of the tongue
        - use tongue blade test – fracture unlikely if patient can bite down on stick hard enough to break it when it is twisted by the examiner
- midface fracture



https://coreem.net/content/uploads/2018/05/Le-Fort-Midface-Fractures-havemursey.com_.png

    - the nasal cavity, paranasal sinuses and orbits act as a series of compartments that progressively collapse and absorb energy, protecting the brain, spinal cord and other structures
    - Le Fort I
        - fracture involving the maxilla at the level of the nasal fossa
        - horizontal plane at the level of the nose
        - palate-facial separation
    - Le Fort II
        - maxilla, nasal bones and medial aspect of orbit involved
            - causes freely mobile, pyramid shaped portion of the maxilla (pyramidal disjunction)

        - fracture line extends from the lower nasal bridge through the medial wall of the orbit, crossing the zygomaticomaxillary process
    - Le Fort III
        - craniofacial disjunction
            - fracture line runs parallel to the base of the skull which separated the midfacial skeleton from the cranium
            - involves the ethmoid bone and cribiform plate at base of skull
        - fracture line extends through the upper nasal bridge and most of the orbit across the zygomatic arch
- temporomandibular joint
    - mechanical impairment may result from condylar or zygomatic arch fracture and can prevent jaw opening (even when paralysed)
- orbital fractures
    - severity can vary
        - oedema and ecchymosis
        - subconjunctival haemorrhage and loss of vision
        - ocular rupture
    - blowout fracture occurs when pressure is applied directly to the eye with fracture of inferior bony structures
        - enophthalmos
        - diplopia
        - impaired eye movement
        - infraorbital hypoaesthesia
- nasal fractures
    - main concerns are epistaxis and septal haematoma
    - septal haematoma



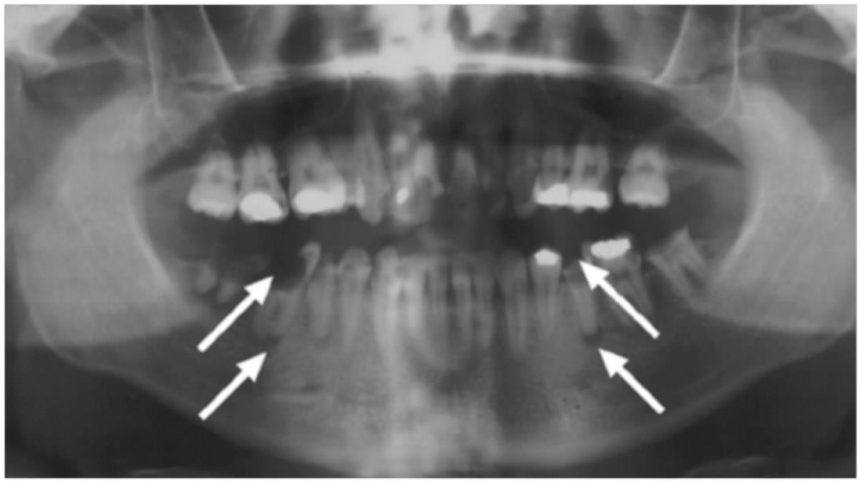
https://www.bmj.com/content/bmj/349/bmj.g6075/F2.large.jpg

        - submucosal vessels are disrupted
        - blood accumulates between perichondrium and cartilaginous septum
        - requires immediate incision and drainage to prevent nasal septum necrosis
            - can lead to saddle nose deformity, nasal septum abscess and perforation
- soft tissue injuries
        - swelling is boggy (septal deviation is firm)
    - abrasions, contusions, lacerations
    - oedema evolving over 24-48 hours can be massive and potentially threaten airway

MaC1 dental abscess

- pathophysiology
    - two most common conditions are caries and periodontitis
        - caries result from acid producing bacteria breaking down the tooth
            - can result in dentine hypersensitivity as fluid moves within exposed dentinal tubules
            - can later cause pulpal exposure, where blood vessels and nerves are exposed to the bacterial pathogens
                - bacteria cause pulpal necrosis and begin to infect the pulpal system, eventually resulting in abscess as they exit via the apical foramen
    - abscesses can spread via tissue spaces, following the path of least resistance
        - can be buccal, sublingual, submandibular, submasseteric or via the pterygomandibular and parapharyngeal spaces
        - submasseteric abscess can result in trismus requiring admission
        - sublingual and submandibular abscess can result in raised floor of mouth and airway risk
            - bilateral sublingual swelling is Ludwig's angina, life threatening condition
        - periodontitis results from oral pathogens releasing toxins, damaging the tooth supporting structures
            - results in receding gums which expose the tooth root surface and cause sensitivity
            - tooth mobility can also cause pain
- history
    - any difficulty with breathing
    - drooling
    - medical history
    - whether they have a dentist
    - any difficulty with mouth opening
    - swelling
    - voice changes
    - restriction of eye opening
    - bad taste (associated with drainage of pus into mouth
- examination
    - raised lymph nodes
    - palpation of abscesses
    - floor of mouth (should be soft, not firm or raised)
    - draining sinuses
    - apply pressure to suspicious teeth – if there is an abscess they will generally be tender
- red flags
    - raised floor of mouth
    - drooling
        - difficulty swallowing and/or talking

    - hot potato voice or alteration to voice
    - difficulty opening eye
    - signs of sepsis
- specific conditions
    - Ludwig's angina
        - rapidly progressive submaxillary, submandibular and sublingual necrotising cellulitis
        - can lead to airway obstruction and death
        - two thirds of cases are odontogenic
        - there is not usually trismus unless there is extension into the parapharyngeal space
        - typically there is dysphagia, neck pain, tooth pain, tongue elevation and/or protrusion and neck swelling
            - there may be signs of airway obstruction such as stridor and dyspnoea
        - patients are often immunosuppressed
        - requires urgent anaesthetic, maxilla-facial and ENT review
            - urgent antibiotics, may require surgical airway and/or surgical drainage
    - erythema associated with abscess spreading down the neck towards the mediastinum
        - sign of rare condition acute purulent mediastinitis
        - infection spreads through the facial planes to the mediastinum
        - life-threatening, requires early diagnosis
            - consider CT
        - early broad spectrum antibiotics
    - Vincent's angina (trench mouth, acute necrotising ulcerative gingivitis)
        - acute febrile pseudo-membranous inflammation of the gingiva
        - more common in immunodeficiency such as malnutrition, HIV
        - usually starts on one side of mouth and spreads, causing gum atrophy, ulceration, enlarged lymph nodes and grey pseudo membrane (may be confused with diphtheria)
        - management is chlorhexidine mouthwash, 3 day course of metronidazole or amoxicillin and improved oral hygiene, plus urgent assessment by a dentist
- investigation
    - orthopantomogram (OPG)
        - helps to assess cause and exclude clinically similar conditions
        - helps identify the causative tooth



https://www.rcemlearning.co.uk/reference/more-dental-emergencies/#1570462240594-2cf163a4-bad7

    - lateral oblique mandible
        - if patient unable to tolerate OPG
        - may be beneficial but not as good as OPG, should only be used as second line
- management
    - refer to general dental practitioner for isolate dental pain
    - maxilla-facial review for those with red flags
        - plus anaesthetic review if concerns about airway risk
    - analgesia
        - ibuprofen first line
        - paracetamol as adjunct or when NSAIDs contraindicated
        - opioids are relatively ineffective for dental pain so should only be used when there is failure of paracetamol and NSAIDs
    - antibiotics
        - should only be given to patients with:
            - symptoms or signs of systemic illness or spreading infection (e.g. cellulitis, lymphadenopathy)
            - high risk patients where complications are likely (e.g. immunocompromise, diabetes)
        - if prescribed, should be amoxicillin or metronidazole for 5 days
            - clarithromycin first line for penicillin allergy
            - consider adding metronidazole or switch to co-amoxiclav if no improvement after 48 hours
        - not generally indicated for otherwise healthy individuals with no signs of spreading infection
            - early source management must be a priority

MaC2 facial wounds

- assessment
    - likelihood of other injuries
        - head/c-spine
        - eyes or teeth
    - wound likely to be contaminated by dirt or foreign bodies

    - injury to deeper structures
        - e.g. tendons, nerves
            - facial nerve
            - parotid/lacrimal ducts
            - medial canthus of eye
    - impairment of blood supply
        - dusky or poorly perfused flap or area of soft tissue distal to wound requires specialist assessment
        - areas of end arterial supply (e.g. tip of nose, ear tips) require special care and avoidance of adrenaline
- management
    - good analgesia
    - sedation may be required for children
    - topical or local anaesthesia or regional block
    - cleaning:
        - superficial wounds
            - clean with saline or aqueous chlorhexidine
        - deep wounds
            - need to be anaesthetised to allow deeper exploration
            - remove foreign bodies/grease
            - irrigate with saline under pressure (through needly on syringe
        - gravel rash (bitumen and dirt ground into skin with skin abrasion)
            - anaesthetise first
            - scrub with brush to remove dirt and prevent tattooing
            - larger areas may require a general anaesthetic
        - ragged wounds
            - trim edges where viability is in doubt
        - glass injuries
            - x-ray if possibility of glass in wound
            - wound exploration if glass present
            - evacuate haematomas as often glass within
- closure
    - non-surgical
        - dressing only
            - for simple lacerations, not gaping or contaminated
            - puncture wounds usually best left open
                - may require exploration or debridement if deep or contaminated
        - tissue adhesive
            - for wounds with clean edges, not requiring suturing and not under tension
            - best for wounds of <3cm with edges easily held together
            - not used on mucosal surfaces
            - if gluing the forehead, pad the eye to avoid dripping
            - hold edges together for 30 seconds after applying glue
            - do not allow glue to enter the wound itself – it acts as a foreign body

- avoid applying too much or applying over active bleeding
- steristrips
  - may be adequate in simple lacerations requiring opposition of slightly separated wound edges
  - should not be used if there is movement or tension
  - should have sufficient space between to allow drainage of fluid and avoid infection
  - keep dry for 72 hours

- surgical
  - scalp
    - bleeding may be profuse
    - usually ceases with firm digital pressure along edges of wound
    - comb hair out of wound (can be helped with Vaseline)
    - 2 layered closure
      - galea: 3/0-5/0 absorbable
      - scalp: 4/0-5/0 nylon
        - removal in 7 days
  - forehead
    - minimal debridement
    - superficial scratches should be cleaned and left to epithelialize (+/- steristrips)
    - sutures: 5/0-6/0 nylon (removal 5-7 days) or Vicryl absorbable
  - cheek
    - check for fractures
      - zygoma, orbital blowout
    - check for involvement of facial nerve and muscle
    - ophthalmology opinion if hyphaema or closed eye with swelling
    - close as for forehead
  - eyelids

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    - if involving the lid margin, refer to ophthalmology
    - look for tarsal plate involvement – refer to ophthalmology
    - simple lacerations can be glued or sutured under tension
      - use 6/0 vicryl absorbable
  - lips

                - accurate approximation of vermillion border and skin required for closure
                - sutures: skin 6/0 nylon (remove in 5 days), mucosa and muscle 4/0 vicryl
                - lacerations of the inner lip rarely need intervention
                - lacerations of the gum margin (e.g. degloving injury) may need dental or max-facs review
            - palate
                - consider possibility of injury to posterior pharynx
                - rarely require suturing unless gaping widely, extending through posterior free margin or continuing to bleed
            - tongue
                - most lacerations do not require suturing
                - if laceration is large, extending through the free edge, full thickness or has ongoing bleeding, plastics opinion is required
            - ear
                - if full thickness involving cartilage, plastics opinion is required

MaC3 post extraction complications

- bleeding
    - lidocaine with adrenaline is commonly used in dental practice, and there can be bleeding once this wears off
    - damp gauze and pressure usually stops the bleeding
    - assessment
        - differentiate between true bleeding socket and blood stained saliva
            - a fresh bleeding socket will be seen to ooze
    - investigations
        - may require FBC and coagulation screen
    - management
        - application of damp gauze
        - application of gauze soaked in tranexamic acid or adrenaline
        - use of haemostatic agents such as hemocollagen or surgicel
            - haemocollagen contains bovine collagen (may be religious or dietary concerns), surgicel is synthetic
        - use of resorbable sutures
            - best place by experienced operators and there is limited space intra-orally for moving instruments around
        - local infiltration of adrenaline (often with local anaesthetic)
        - review of anticoagulation regimes with risk-benefit discussion
            - consider reversal
        - devices such as bipolar machines
            - not normally required
        - consider use of 5% tranexamic acid mouthrinse 4 times a day as prophylaxis afterwards
        - do not advise rinsing with water or saline due to risk of dislodging further clot

- patient should be advised to restart saltwater rinse the following day after a stable clot has formed to prevent infection

- dry socket
    - aetiology unknown – three theories
- blood clot initially present is dislodged and lost and the exposed bone infected by microorganisms
- blood clot undergoes fibrinogenesis as a result of the healing and remodelling process
- bacterial enzymatic processes implicated in the breakdown of the blood clot
    - risk factors include age, difficult extraction and previous infection
    - assessment
        - usually presents with a bad taste and sometimes a bad odour
        - tender region with denuded bone and food debris are suggestive
    - investigation
        - not normally required
        - x-ray may be done to look for tooth fragments in the socket
    - management
        - irrigation of the area with saline
        - aveogyl, a dressing material containing eugenol, which helps relieve pain and aid healing
        - avoid chlorhexidine as direct socket irritant – has resulted in 2 deaths in the UK due to previously unknown allergy
        - avoid antibiotics unless signs of spreading infection, systemic infections or immunocompromised patients

MaC4 TMJ dislocation

- anatomy
    - the mandible is a u shaped bone comprising two hemimandibles completely fused at the symphysis by the age of two years
    - the mandible articulates with the mandibular fossa of the temporal bone forming the temporomandibular joints
    - the TMJ is split into two sections by an articular disc (or meniscus), a fibrocartilaginous structure enabling a greater range of movement of the joint
- mechanism
    - dislocation may be unilateral or bilateral and occur in anterior, posterior, lateral and superior positions
        - anterior is the most common
        - the others are associated with fracture to the mandible or base of skull
    - in trauma it is usually caused by direct downward force to a partially opened mouth
    - in predisposed patients with shallow mandibular fossae or underdeveloped mandibular condyles, certain repeated activities may initially sublux, then dislocate, the mandible
    - most common mechanism relates to excessive opening of the mouth due to:
        - yawning
        - laughing
        - shouting

        - eating
        - during dental work
    - connective tissue disorders such as Marfan's or Ehlers-Danlos syndrome increase the likelihood of dislocation
    - once the mandible has dislocated anteriorly, spasm of the masseter and pterygoid muscles occurs, which further trap the dislocated condyle
- clinical features
    - inability to close mouth
    - difficulty speaking or swallowing
    - malocclusion
    - pain localised anterior to the tragus
    - prominent-appearing lower jaw
    - preauricular depression
    - in posterior dislocation, blood in ear
        - must examine auditory canal
        - tympanic plate of temporal bone can be fractured and tear the auditory canal membrane
- management
    - x-ray – usually OPG
    - may be easier to reduce one side at a time
    - syringe reduction technique
        - ask patient to place an empty 5 or 10ml syringe between the upper and lower molars on one side of the mouth
        - direct patient to roll syringe back and forth until reduction achieved
        - repeat on opposite side unless reduction spontaneously achieved
    - extra-oral reduction
        - patient in seated position
        - thumb on zygomatic arch, fingers of same hand behind mandible
        - on opposite side, place thumb on coronoid process of mandible
        - use fingers of first hand to pull mandible forward while using zygomatic arch to brace
        - firm and consistent pressure on coronoid process with other hand
        - once first side is reduced, apply same technique on other side
    - standard intraoral technique is designed to push the mandible inferiorly and posteriorly back into the mandibular fossa
        - can be done from an anterior or posterior approach
        - it is important to wear strong gloves and position the thumbs behind the last molars to protect against human bites when the mouth snaps closed due to muscle spasm
            - a bite block may also be used
    - intra-oral wrist pivot method
        - patient in seated position
        - face the patient and grasp mandible with thumbs at apex of the mentum
        - place well-wrapped and gloved fingers on the occlusal surface of the inferior molars
        - apply cephalad force with thumbs and caudal force with fingers
        - pivot the wrists to reduce the joint
    - analgesia/sedation

        - opioid and a sedative agent such as midazolam is common
        - propofol is sometimes used
    - posterior, medial or lateral dislocations
        - usually associated with fractures and should be referred to a maxillofacial surgeon
- post-reduction
    - check x-ray
        - confirm position and exclude fracture
    - discharge advice
        - soft diet for first few days to minimise stress on TMJ
        - avoid wide mouth opening for two weeks and support jaw if laughing or yawning
            - encircling bandage is unnecessary unless patient cannot understand or comply with instructions
    - max-facs follow up

MENTAL HEALTH

MHP1 aggressive or disturbed behaviour

- background
    - aggressive or violent behaviour may represent a serious underlying mental health condition
    - behaviour may be impulsive (occurs due to a trigger) or proactive (premeditated and well thought out)
    - issues causing stress in general in the ED include
        - illness/injury
        - pain
        - stress
        - parking difficulties
        - not being able to smoke
        - being asked not to use mobile phones
        - frustration over waiting times
    - most patients respond positively to good customer care and courteous communication
        - provision of appropriate, timely and accurate information may prevent initiation or escalation of aggressive behaviour
    - threats to and assaults on staff have been shown to be associated with staff sickness, absence, turnover, loss of productivity, loss of commitment, fear, anxiety, loss of confidence
- aggression
    - the average physical fight between two individuals lasts for only 7 seconds
        - up to 15 violent acts may be exchanged in this time
        - injury tends to occur in the first 3 seconds
    - because incidents happen so quickly, it is not possible to rely on panic buttons or security staff in the ED

    - aggressive or violent behaviour may occur due to many underlying medical, toxological or mental problems, which need to be recognised and treated where needed
    - reversible causes should be identified during the initial evaluation
        - the mnemonic FIND ME may be helpful:
            - **F**unctional
            - **I**nfectious
            - **N**eurological
            - **D**rugs
            - **M**etabolic
            - **E**ndocrine
    - bedside blood glucose and oxygen saturations should be checked
    - possible causes include:
        - medical
            - hyperthyroidism
            - hypoglycaemia
            - traumatic brain injury
            - delirium
        - psychiatric
            - depression
            - conduct disorder
            - attention deficit hyperactivity disorder
            - bipolar disorder
            - impulse control disorder
            - psychosis
            - impulsive behaviour
            - substance intoxication/withdrawal
            - post traumatic stress disorder
- legal principles
    - the Health and Safety at Work Act
        - requires that workers and other persons should be provided with the highest level of protection that is reasonably practical against harm to their health, safety and welfare
    - what the law allows
        - the law allows the use of reasonable force to protect ourselves and others in certain circumstances
    - the Human Rights Act (1998)
        - determines that any use of force is not only reasonable but also proportionate and necessary, as determined by the person at the time
- assessment
    - subject's behaviour
        - aggressive or violent behaviour covers a spectrum of behaviours:
            - calm and non-threatening
                - frustration only without overt signs of agitation
            - verbal agitation
                - change in speech patterns
            - verbal hostility

                - more aggression is suggested
            - verbally threatening
                - actual threats are conveyed
            - physically threatening
                - patient assumes a fighter's stance and makes a fist
            - physically violent
                - patient physically attacks
    - subject's behaviour and increasing level of threat
        - compliant/non-threatening
            - complies with staff request
        - verbal and non-verbal indicators of threat/violence
            - warning and danger signs:
                - warning signs – communication may help to de-escalate:
                    - shouting, loud voice
                    - angry demeanour
                    - tense posture, holding arm rails tightly, clenching fists
                    - pacing, frequent change in body position
                    - aggressive behaviour, pounding walls, throwing objects, hitting or kicking
                - danger signs (usually herald imminent physical threat) – communication unlikely to help, exit or perform physical intervention:
                    - fist clenching
                    - facial pallor
                    - lips tightening over bared teeth
                    - eyebrows and chin drop
                    - hands above the waist
                    - target-acquisition glances (looking at a body part as a prelude to striking that area)
        - active aggression
            - subject strikes
        - assaultive/aggression/violence
            - repeated and focused attempts at assault
        - serious/aggravated violence
            - production of a weapon with the intent to use it in an assault
- impact factors (render a situation more or less dangerous)
    - gender/age/size/strength
        - relative size compared to yours
    - previous knowledge/history
        - someone who has been violent before is more likely to be violent again
    - alcohol/drugs
        - reduces the ability to reason
        - may cause loss of control making an assault more likely
    - special skills
        - a martial arts practitioner or boxer will know how to respond more efficiently and with greater effect

- imminent danger
  - behaviour indicating imminent assault
- position of disadvantage
  - no route of escape, or a barrier between you and the aggressor
- environment
  - difficulties in access/egress, no other support available
- weapons
  - production of weapons or availability of weapons of opportunity

- management
  - response options
    - disengage
      - depart and call for help
      - put barriers between you and the patient
      - best option for any serious threat
    - communication strategies
      - verbally de-escalate the situation with communication skills
        - LEAPS
          - **l**isten
            - ask open questions such as 'what seems to be the problem?'
            - listen actively and let people have their say
            - do not interrupt
            - remain objective
          - **e**mpathise
            - indicate empathy even if you do not agree
            - e.g. 'that sounds terrible', 'I see'
          - **a**sk
            - question them to clarify their concerns
          - **p**araphrase
            - 'reading back' key parts of their concerns shows that you are listening and have engaged with them
          - **s**ummarise
            - summarise their concerns and try developing a course of action
    - tips and ground rules
      - create a calm, polite, respectful atmosphere
      - respect personal space
        - maintain a safe distance (at least two arm lengths) and provide space for an easy exit
      - avoid touching an angry or agitated person
      - stay on the same physical level, do not look down on them; avoid sudden movements
      - keep posture neutral and maintain a non-confrontational body posture
      - do not stare at the patient; eye contact should convey sincerity
    - establish verbal contact in a calm clear voice

        - use concise and simple language
            - e.g. ‘how can we help you?’
- active listening
    - restate what the patient has said
    - may help to improve mutual understanding (e.g. ‘tell me whether I have this right’)
- acknowledge their frustration
- align goals
    - emphasise common interests
    - focus on the big picture
    - find ways to make small concessions
- monitoring
    - be aware of progress
    - known when to disengage
    - do not insist on having the last word
    - have a staff member sit with the patient
- agree, or agree to disagree
- refer to rules of common decency and set clear limits – inform the patient that violence or abuse cannot be tolerated
- offer alternate choices if possible to give them choice in the outcome
- debrief the patient and staff
- if security need to be activated
    - they should gather outside the door or close by, within eye contact of the room
    - a strong show of force may calm a potentially violent patient without the need for restraint
    - directly address the issue of violence
- inquire about
    - suicidal or homicidal ideation or plan
    - possession of weapons
        - their belongings should be searched
    - current use of medications
    - acknowledge the obvious (e.g. ‘you look angry’)
    - if they become more agitated it may be helpful to speak in a conciliatory manner (e.g. you obviously have a lot of willpower and are good at controlling yourself’)
- assertions using different communication skills
    - for people displaying warning signs without danger signs (signs of imminent violence) we may need to escalate our communication strategy to assertion
    - examples:
        - ‘I want to help you, but I need you to stop shouting/swearing’
        - ‘this is what we can offer you’
        - ‘if you remain calm we will be able to help you’
        - ‘you tell me what it is that you want to do’

        - when no other options remain, a reasonable, proportionate and necessary physical response should be used if there is a need to establish control or prevent harm to anyone
            - we should adopt a stance which affords minimal body exposure, offers some protection and shows a readiness to communicate
    - the fighting arc
        - an assault can only be delivered within the fighting arc – the zone in which a fist or kick can be delivered
        - we should conduct communications with a potentially challenging subject outside the fighting arc
    - lawful physical intervention
        - to be lawful, it must be used appropriately and in context
        - physical skills include breakaway skills, blocks, pre-emptive strikes and restraint
        - physical skills can be used:
            - to defend yourself and protect others from immediate threat
            - to restrain an agitated and at-risk individual
        - appropriate training is an advantage
        - physical skills training needs to include the risks of restraint, including positional asphyxia and excited delirium
    - restraint
        - behaviour posing a safety risk should be managed before a medical and psychiatric examination can be performed
        - indications for emergency sedation and restraint include prevention of imminent harm to the patient, to others or to the immediate environment
        - ideally there should be a predetermined ED protocol that can be implemented
        - restraint must be carefully applied in order to minimise restrictions on chest wall movement and ensure no negative impact on respiration or other vital functions
        - practical training is recommended
    - excited delirium
        - individuals display bizarre and aggressive behaviour, often associated with drug use
        - they may feel no pain, be abnormally strong and may rip off their clothing
        - they are frequently brought into hospital by the police
        - they are at great risk because they are:
            - hyperpyrexial
            - dehydrated
            - acidotic
            - hypoglycaemic
            - fatigued
        - it is essential to intervene rapidly to prevent physical or mental deterioration – restraint and rapid tranquilisation will be needed
    - rapid tranquilisation
        - warranted for patients who are:
            - without capacity

                - agitated
                - a risk to themselves
                - a risk to others
            - for most patients, 2mg lorazepam PO is indicated
            - when urgent control is required, IM haloperidol (5-10mg) and/or IM lorazepam 2mg
                - appropriate monitoring is required
                - IM drugs can take up to 30 minutes to have an appreciable effect
                - IV drugs should only be used in exceptional circumstances
- investigations
    - restraint may be required to facilitate investigations
    - background information is helpful and may have to be obtained from a variety of sources, e.g. hospital records, GP, family and friends
    - history of drug use, prescribed and illicit
    - vital signs and blood glucose
    - blood gas when safe when excited delirium suspected
    - further investigation depending on initial information and observation
- pitfalls
    - failure to recognise medical causes for behaviour such as hypoxia or head injury
        - acute changes in behaviour are a medical condition until proven otherwise
    - failure to disengage when danger signs are present
    - dismissing challenging patients from the department before excluding significant medical or psychiatric causes

MHP2 anxiety/panic

- generalised anxiety disorder
    - risk factors
        - age 35-54
        - being divorced or separated
        - living alone or as a lone parent
    - protective factors include:
        - age between 16 and 24
        - being married or cohabiting
    - diagnostic criteria (ICD-10)
        - excessive anxiety and worry (apprehensive expectation) occurring more days than not for at least 6 months, about a wide range of events or activities (such as work or school performance)
        - the person finds it difficult to control the worry
        - the anxiety or worry are associated with (at least some symptoms present more days than not for the preceding six months) three or more (only one for children) of:
            - restlessness or feeling keyed up or on edge
            - being easily fatigued
            - difficulty concentrating or mind going blank
            - irritability
            - muscle tension
            - sleep disturbance

        - anxiety and worry owing to panic disorder, social phobia, obsessive-compulsive disorder and separation anxiety disorder are excluded
        - at least four of the following must also be present (with at least one from the first group):
            - autonomic arousal symptoms
                - palpitations or pounding heart
                - accelerated heart rate
                - sweating
                - trembling or shaking
                - dry mouth (not due to medication or dehydration)
            - symptoms involving chest and abdomen
                - difficulty breathing
                - feeling of choking
                - chest pain or discomfort
                - nausea or abdominal distress (such as churning in the stomach)
            - symptoms involving mental state
                - feeling dizzy, unsteady, faint or light-headed
                - feeling that objects are unreal (derealisation) or that the self is 'not really here' (depersonalisation)
                - feeling of losing control, 'going crazy' or passing out
                - fear of dying
            - general symptoms
                - hot flushes or cold chills
                - numbness or tingling sensations
                - muscle tension or aches and pains
                - restlessness and inability to relax
                - feeling keyed up, on edge or mentally tense
                - a sensation of a lump in the throat or difficulty in swallowing
            - other non-specific symptoms
                - exaggerated response to minor surprises or to being startled
                - difficulty in concentrating or mind going blank because of worrying or anxiety
                - persistent irritability
                - difficulty in getting to sleep because of worrying
    - management (NICE):
        - identify the diagnosis and inform the patient as soon as possible so treatment can be commenced
        - the patient's preference for method of treatment
        - independent interpreters if necessary
        - emphasise to the patient that the disease can be managed and give as much information as possible including written information and access to self-help and psycho-educational groups
        - make an assessment of the patient's functional disability and distress
        - assess past experience and response to previous treatment
        - in terms of long term effectiveness, the best results are from psychotherapy, followed by medication, followed by self-help
        - patients should be provided with contact details in case of crisis

- psychological therapy
  - CBT is the technique of choice
    - optimum duration is 16-20 hours over 4 months
  - anxiety management treatment
    - structured therapy involving education, relaxation, training and exposure
- pharmacological interventions
  - considerations:
    - age of patient
    - previous treatment response
    - risks of deliberate self-harm or accidental overdose
    - tolerability
    - possible interactions with existing medication
    - patient preference
    - cost, where there is equal effectiveness
  - where a rapid response is required:
    - sedating antihistamines or benzodiazepines may be helpful
      - benzodiazepines should not be used beyond four weeks
  - antidepressants
    - good at relieving anxiety even where there is no depression
    - take longer to work than benzodiazepines but can be used for longer
    - NICE recommends an SSRI or venlafaxine as the first choice
      - if one is not suitable or there is no improvement after a 12 week course, another should be offered
      - NICE recommends sertraline first line
    - patients should be informed about:
      - potential side effects (including transient increase in anxiety at the start of treatment)
      - possible discontinuation/withdrawal symptoms
      - delay in onset of effect
      - time course of treatment
      - need to take medication as prescribed
    - pregabalin should be considered in patients who cannot tolerate SSRIs
    - duloxetine can be helpful for anxiety
    - there is no evidence to guide duration of treatment
  - self-help
    - can be effect but best used as part of a stepped-care approach
    - there is some evidence for internet based CBT
- prognosis
  - is it usually a chronic disease that is controlled rather than cured
  - chronic anxiety conditions are associated with increased morbidity

- panic disorder
  - definition

        - panic attacks must be associated with >1 month's duration of subsequent, persisting anxiety about recurrence of the attacks, the consequences of the attacks or significant behavioural changes associated with them
        - a panic attack is defined as a discrete episode of intense subjective fear, where at least four of the following symptoms arise rapidly and peak within ten minutes
            - palpitations, pounding heart or accelerated heart rate
            - sweating
            - trembling or shaking
            - dry mouth
            - feeling short of breath, or a sensation of smothering
            - feeling of choking
            - chest pain or discomfort
            - nausea or abdominal distress
            - feeling dizzy, unsteady, lightheaded or faint
            - derealisation or depersonalisation
            - fear of losing control or going crazy
            - fear of dying
            - numbness or tingling sensations
            - chills or hot flushes
        - attacks usually last at least 10 minutes but duration is variable
        - symptoms must not arise as a result of alcohol or substance misuse, medical conditions or other psychiatric disorders in order to satisfy the diagnostic criteria
    - management
        - recognition and diagnosis
        - general treatment
            - involve the family or carer if the patient allows
            - avoid anxiety =-inducing substances such as caffeine
            - treat alcohol or drug misuse if indicated
        - CBT
            - recognition of factors which trigger the panic and behavioural methods to cope with the symptoms
            - usually 1-2 hours a week over 4 months
        - medication
            - antidepressants (SSRIs as first line)
            - use of self-completed questionnaires to monitor response to treatment
        - self-help
            - books based on CBT principles, support groups
            - promotion of exercise
            - advice on how to control symptoms with abdominal/diaphragmatic breathing
    - prognosis
        - unclear, may last for years

MHP3 physical symptoms unexplained by organic disease

- definition of medically unexplained symptoms
    - repeated medical help-seeking for multiple medical symptoms without organic disease
    - physical symptoms for which no clear or consistent organic pathology can be demonstrated
- aetiology
    - causative factors are similar to those for anxiety and depression
        - stress at home or work, history of childhood or family illness and high deprivation index may all play a part
        - a history of past or recent abuse is often associated
            - in the case of chronic pelvic pain, around a third of patients have some history of abuse
- epidemiology
    - 25-50% of primary care patients have MUS
- presenting features
    - wide spectrum of complaints ranging from mild transitory illness to chronic disorders with severe disability
    - more likely if there is a past or current history of depression or anxiety
    - many patients with depression present exclusively with somatic symptoms
    - patients do not fit the biomedical model that focuses solely on exclusion of disease
        - however the exclusion of physical disease may not cure the patient, who may still seek medical care
- management
    - should be co-ordinated by primary care with the whole team aware so a consistent approach is applied
    - physical exercise seems to be of benefit
    - the importance of pleasurable private time should be emphasised
    - making a diagnosis
        - combines consideration of MUS and classical psychiatric disorders, and exclusion of serious physical problems
        - which tests are done depends on the symptoms
        - a psychological evaluation should also be carried out, but evidence of a psychiatric disorder does not rule out somatisation
        - doctors may test for and even treat non-existent organic illness
            - there is a high cost of use of services, unnecessary laboratory testing, increased costs and high iatrogenic complication rate
    - psychotherapy
        - approaches derived from cognitive behavioural therapy have been shown to reduce the intensity and frequency of somatic complaints and can improve functioning
        - the treatment starts with the mutual agreement that whatever the patient has been thinking and doing about the condition has not been successful
        - it then begins to challenge the patient's beliefs and maladaptive behaviours
        - mindfulness based cognitive therapy may be helpful
    - reattribution model (Goldberg and Gask)
        - key principles are:

                - to make the patient feel understood
                - to broaden the agenda
                - to negotiate a new understanding of the symptoms, including psychosocial factors
        - communication
            - different communication skills to usual may be required
            - if reassurance does not address patients' specific concerns it may exacerbate their presentation of somatic symptoms
            - effective explanations provide real mechanisms for understanding, based on patients' concerns, often linking physical and psychological factors
        - pharmacotherapy
            - antidepressants have been reported to have some effect in MUS, possibly due to the treatment of comorbid depression

MHP4 self-harm

- background
    - self-harm refers to an intentional act of self-poisoning or self-injury, irrespective of the motivation or apparent purpose of the act
    - it is an expression of emotional distress
    - deliberate self-harm may be caused by:
        - a behaviour (e.g. cutting) intended to cause self-harm
        - ingesting a substance in excess of the prescribed or generally recognised therapeutic dose
        - ingesting a recreational or illicit drug that was an act that the person regarded as self-harm
        - ingesting a non-ingestible substance or object
    - it is not an attempt at suicide in the majority of cases
    - it is usually an attempt to maintain control in very stressful circumstances
    - it is usually done in private and hidden from anyone else
- epidemiology
    - common, with a lifetime prevalence of at least 5-6%
    - more common among younger people
    - increases the likelihood that a person will eventually die by suicide 50-100-fold above the rest of the population in a 12 month period
    - associated with a wide range of psychiatric problems
    - other risk factors include victims of domestic violence, socio-economic disadvantage and those with eating disorders
- management
    - drug overdose
        - activated charcoal if indicated (usually within one hour)
            - toxins not bound to charcoal include hydrocarbons, alcohols (methanol, ethanol, ethylene glycol), metals (lithium, iron, potassium, lead, silver, mercury), malathion, corrosives
        - manage as per TOXBASE advice
    - later management
        - patients should be fully involved in decision-making
        - integrated comprehensive psychosocial assessment of needs and risks

        - people often do not know why they self harm
        - assessment of risk including:
            - main clinical and demographic features know to be associated with risk of further self-harm and/or suicide
            - identification of the key psychological characteristics associated with risk, especially depression, hopelessness and continuing suicidal intent
                - indicators of suicidal risk include strong suicidal intent, high lethality, precautions against being discovered, psychiatric illness
        - psychological intervention
- prognosis
    - risk of repetition of self-harm and of later suicide is high
        - more than 5% of people who have been seen at a hospital after self-harm will have committed suicide within 9 years
        - some young people self-harm on a regular basis, whilst others do it just once or twice
        - for some people it is part of coping with a specific problem and they step when the problem is resolved
        - the main risk factor for repetition is long-standing psychological vulnerabilities
        - physical health and life expectancy are severely compromised in people who self-harm

MHP5 refusal of treatment

- GMC personal beliefs guidance
    - 'you must respect a competent patient's decision to refuse an investigation or treatment, even if you think their decision is wrong or irrational; you may advise the patient of your clinical decision but you must not put pressure on them to accept your advice; you must be careful that your words and actions do not imply judgement of the patient or their beliefs or values'
    - 'if the patient is a child who lacks capacity to make a decision, and both parents refuse treatment on the grounds of their religious or moral beliefs, you must discuss their concerns and look for treatment options that will accommodate their beliefs; you should involve the child in a way appropriate to their age and maturity; if following a discussion of all the options you cannot reach an agreement, and treatment is essential to preserve life or prevent serious deterioration in health, you should seek advice on approaching the court'
    - 'in an emergency, you can provide treatment that is necessary to save life or prevent deterioration in health without consent or, in exceptional circumstances, against the wishes of a person with parental responsibility
- GMC ethical guidance
    - if you disagree with a patient's choice of option
        - 'you must respect your patient's right to decide; if their choice of option (or decision to take no action) seems out of character or inconsistent with their beliefs and values, it may be reasonable to check their understanding of the relevant information and their expectations about the likely outcome of this

option and reasonable alternatives; if it's not clear whether a patient understands the consequences of their decision, you should offer more support to help them understand the relevant information; but you must not assume a patient lacks capacity simply because they make a decision that you consider unwise'
        - 'if a patient asks for a treatment or care that you don't think would be in their clinical interest, you should explore their reasons for requesting it, their understanding of what it would involve, and their expectations about the likely outcome; this discussion will help you take account of factors that are significant to the patient and assess whether providing the treatment or care could serve the patient's needs; if after discussion you still consider that the treatment or care would not serve the patient's needs, then you should not provide it; but you should explain your reasons to the patient and explore other options that might be available, including their right to seek a second opinion'
    - treatment in emergencies
        - 'in an emergency, decision may have to made quickly, so there'll be less time to apply this guidance in detail, but the principles remain the same; you must presume a conscious patient has capacity to make decisions and seek consent before providing treatment or care'
        - 'in an emergency, if a patient is unconscious or you otherwise conclude that they lack capacity and it's not possible to find out their wishes, you can provide treatment that is immediately necessary to save their life or to prevent a serious deterioration of their condition; if there is more than one option, the treatment you provide should be the least restrictive of the patient's rights and freedoms, including their future choices'
        - '[…] if the patient regains capacity while in your care, you must tell them what has been done and why, as soon as they are sufficiently recovered to understand; and you must discuss with them the options for any ongoing treatment'
    - mental capacity
        - 'capacity is the ability to make a decision; this ability can vary depending on a patient's condition and how it changes over time, and on the nature of the decision to be made; for this reason, capacity is described as decision-specific and time-specific; so, a person can only have capacity or lack capacity to make a specific decision at a specific time'
    - presuming capacity
        - 'you must start from the presumption that every adult patient has capacity to make decisions about their treatment and care […]'
    - 'assessing capacity
        - 'a person has capacity if they can do all the following:
            - understand information relevant to the decision in question
            - retain that information
            - use the information to make their decision
            - communicate a decision'
        - 'if you believe that a patient may lack capacity to make a decision, you must assess their capacity using the test set out in the relevant legislation, taking account of the advice in the relevant guidance; if you find it difficult to judge

whether a patient has capacity to make a decision, you should seek support from someone who knows the patient well, for example another member of the healthcare team or someone close to the patient'

MHC1 alcohol and substance misuse

- RCEM guidance: drug misuse and the Emergency Department
    - summary of recommendations
        - patients attending the Emergency Department with presentations that might possibly be related to drug use, or may be a marker for drug use, should be asked about this as part of their clinical assessment
        - patients with known mental illness or presenting with mental health problems (e.g. overdose) should be asked about illicit drug use, particularly those with psychosis
        - young people/adolescents should be asked about illicit drug use in a manner and environment that supports disclosure
        - patients presenting with a drug misuse issue or who are known to use illicit drugs should receive written advice signposting them to a drug service
        - use of Mental Health Liaison teams or alcohol liaison nurses to provide advice and support for patients whilst in the ED is encouraged if resource allows
        - brief interventions in the ED for illicit drug use are of unproven benefit and not currently recommended
        - drug detoxification should not be undertaken by the ED, but clinicians should know how to treat acute intoxication or withdrawal
        - EDs are recommended to have clear guidance in place with regard to patients requesting drug detoxification, methadone and any short-term interventions the ED may or may not be involved with, for example supply of benzodiazepines or buprenorphine for withdrawal symptoms
        - EDs are recommended to have links to the local substance misuse service either via an alcohol nurse, drug and alcohol liaison team (DALT) or mental health liaison service, to refer new patients but also to confirm existing maintenance prescriptions which can usually be done via pharmacists contacting community dispensing pharmacies
        - urine toxicology screens are not recommended, they are expensive, prone to misleading results and rarely influence immediate management decisions
        - consider reporting any adverse reactions to Novel Psychoactive Substances (NPS) to the RIDR scheme (Reporting Illicit Drug Reactions) on the Public Health England website
        - all drug related deaths should be reported to the coroner
- example of an adolescent screening tool (CRAFFT questionnaire)
    - have you ever ridden in a **c**ar driven by someone (including yourself) who was high or had been using alcohol or drugs?
    - do you ever use alcohol or drugs to **r**elax, feel better about yourself or fit in?
    - do you ever use alcohol or drugs while you are by yourself, **a**lone?
    - do you ever **f**orget things you did while using alcohol or drugs?
    - do your family or **f**riends ever tell you that you should cut down on your drinking or drug use?
    - have you ever got into **t**rouble whilst you were using alcohol or drugs?

- drug classification
    - A
        - cocaine
        - ecstasy
        - hallucinogens (LSD, magic mushrooms)
        - opiates
    - A/B
        - amphetamine/methamphetamine
    - B
        - cannabis
        - ketamine
        - mephedrone
    - B/C
        - tranquilisers
    - C
        - anabolic steroids
    - New Psychoactive Substances (NPS)
        - drugs designed to replicate the effects of illegal substances whilst remaining legal
        - four main categories:
            - synthetic cannabinoids
                - mimic cannabis
                - e.g. black mamba, spice
                - the chemicals blended into the base plant matter act on the brain in a similar way to cannabis, but they bear no relation to the cannabis plant
            - stimulant-type drugs
                - mimic substances such as amphetamine, cocaine and ecstasy
                - include mephedrone, BZP, ethylphenidate
            - 'downer'/tranquiliser type drugs
                - mimic tranquiliser or anti-anxiety drugs
                - include etizolam, pyrazolam, flubromazepam
            - hallucinogenic drugs
                - mimic substances like LSD
                - possession for personal use is not illegal, but it is illegal to produce, supply or import them for human consumption
- DSM-5 criteria for substance abuse disorders
    - ten separate classes of drugs
        - alcohol
        - caffeine
        - cannabis
        - hallucinogens
        - inhalants
        - opioids
        - sedatives/hypnotics/anxiolytics
        - stimulants

        - tobacco
        - other unknown substances
    - a problematic pattern of use leading to clinically significant impairment or distress is manifested by two or more of the following within a 12 month period:
        - often taken in larger amounts or over a longer period than was intended
        - a persistent desire or unsuccessful efforts to cut down or control use
        - a great deal of time is spent in activities necessary to obtain, use or recover from the substance's effects
        - craving or a strong desire or urge to use the substance
        - recurrent use resulting a failure to fulfil major role obligations at work, school or home
        - continued use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by its effects
        - important social, occupational or recreational activities are given up or reduced because of use
        - recurrent use in situations in which it is physically hazardous
        - continued use despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance
        - tolerance
        - withdrawal
    - severity
        - mild – 2-3 criteria
        - moderate – 4-5 criteria
        - severe – 6 or more criteria
- RCEM alcohol toolkit
    - aims
        - promotion of best practice in the area of alcohol management
        - advancement of safe and effective care in this area
        - education and training of emergency medicine doctors
    - operational aims
        - identification and brief advice
        - departmental alcohol care team
        - alcohol champions
        - tackle re-attenders
    - strategic management
        - early intervention is important
        - people who need support for alcohol problems should be routinely referred to specialist alcohol services for comprehensive assessment and appropriate treatment
        - every acute hospital should have a specialist MDT alcohol care team led by an 'alcohol champion', tasked with meeting the needs of those attending with alcohol-related problems and preventing readmissions
    - screening tools
        - include the Paddington Alcohol Test (PAT), the Fast Alcohol Screen Test (FAST) and the AUDIT C tool
            - AUDIT C is a shortened version of the full AUDIT tool

            - it identifies people who are drinking at increasing/higher risk drinking before their drinking becomes problematic or dependent
            - it can be used with patients of all ages and is recommended by NICE
        - the alcohol champion should decide which ED staff should be doing the screening, and on which patient groups
- alcohol nurse and brief interventions
    - it is recommended that there is at least one alcohol and drug nurse specialist in each ED
        - they should be separate to the nurse for hospital ward referrals
        - NHS England recommends a four nurse service to provide cover from 7/8am to 10pm 7 days a week
    - brief intervention can be:
        - a sentence or two of feedback to the patient about his/her drinking based on the screening tool and the person's circumstances
        - a sentence or two of feedback plus and information leaflet
        - five minutes of advice based on the FRAMES structure (feedback, responsibility, advice, menu of options, empathy, self-efficacy)
    - the alcohol nurse should be available for any patients presenting with an alcohol related illness or injury and be able to identify them from the computer system without direct referral
- clinical decision units
    - can be used to allow patients to 'sober up' and have screening and brief intervention
    - thiamine should be given 3 times a day
        - 2 x I+II (4 ampoules)
    - patients with withdrawal can also be managed in a CDU with a chlordiazepoxide regimen and CIWA (Clinical Institute Withdrawal Assessment for Alcohol) scoring
- management of the intoxicated patient
    - patients should be managed in a safe place (majors or CDU) and not left in the waiting area to sober up, as they are high risk patients
- reattenders
    - care plans
        - should be written by the alcohol specialist nursing team and the ED alcohol consultant champion
        - they can provide alerts and the details of key personnel involved in the patient's care
- paediatric population
    - physical and psychological risks are much greater for children and young people
    - childhood drinking increases the likelihood of risky behaviour such as risk of being assaulted, underage pregnancy, contracting sexually transmitted disease and becoming alcohol dependent
        - it increases the risk of being involved in criminal and anti-social behaviour and reduces potential at school

                - child safeguarding applies to under 18s, and there should be a policy for safeguarding those between 16 and 18 who will not be admitted under paediatrics
        - scoring should be done 4 hourly routinely, two hourly if greater than 6 and hourly if greater than 9
        - scoring for alcohol consumption includes:
            - temperature
            - pulse
            - respiration rate
            - tremor (arms extended, fingers spread)
            - sweating (observation)
            - clouding of sensorium (ask about day and place)
            - quality of contact
            - agitation
            - thought disturbances (flight of ideas, paranoia)
            - visual disturbances (photophobia, hallucination)

MHC2 depression

- background
    - refers to both negative affect (low mood) and/or absence of positive affect (loss of interest and pleasure in most activities)
    - usually accompanied by a variety of emotional, cognitive, physical and behavioural symptoms
    - most common psychiatric disorder, carries a high burden in terms of treatment costs, effect on families and carers and loss of workplace productivity
    - WHO leading cause of disability globally
    - may become chronic, particularly if inadequately treated
    - more than 80% are seen in primary care, those seen in secondary care are skewed towards severe disease
    - annually, 5% of adults have an episode of depression
        - one in four women and one in five men develop depression severe enough to require treatment at some time in their lives
- diagnosis (DSM-5)
    - diagnosis of major depression requires at least one of:
        - persistent sadness or low mood nearly every day
        - loss of interest or pleasure in most activities
    - plus at least three or four of (minimum 5 in total):
        - fatigue or loss of energy
        - worthlessness, excessive or inappropriate guilt
        - recurrent thoughts of death, suicidal thoughts or actual suicide attempts
        - diminished ability to think/concentrate or increased indecision
        - psychomotor agitation or retardation
        - insomnia/hypersomnia
        - changes in appetite and/or weight loss
    - symptoms should have been present persistently for at least two weeks and must have caused clinically significant distress and impairment

        - they should not be due to a physical/organic factor (e.g. substance abuse) or illness
    - severity (based on extent of symptoms and their functional impact):
        - subthreshold - <5 symptoms
        - mild depression – few, if any symptoms in excess of the 5 required to make the diagnosis, with symptoms resulting only in minor functional impairment
        - moderate depression – symptoms or functional impairment are between mild and severe
        - severe depression – most symptoms present and the symptoms markedly interfere with normal function; can occur with or without psychotic symptoms
- risk factors
    - female gender
    - past history of depression
    - significant physical illnesses, particularly those causing disability or chronic pain
    - other mental health problems, such as schizophrenia or dementia
    - psychosocial problems – e.g. divorce, unemployment, poverty
    - risk factors for depression in young people and children include:
        - family discord
        - bullying
        - physical, sexual or emotional abuse
        - comorbid disorders including drug and alcohol use
        - history of parental depression
        - ethnic and cultural factors
        - homelessness
        - refugee status
        - living in institutional settings
- screening
    - NICE encourages a two question approach for at-risk groups:
        - during the past month, have you:
            - felt low, depressed or hopeless?
            - had little interest or pleasure in doing things
        - a positive answer should trigger a further assessment, but a negative answer does not exclude depression
- assessment
    - self-report symptom scales are widely used and include:
        - the Patient Health Questionnaire (PHQ-9)
        - the Hospital Anxiety and Depression (HAD) scale (not available digitally and must be purchased)
        - Beck's depression inventory II
    - an individual considered to have depression should be fully assessed, including:
        - full history and examination, including mental state examination
            - consider organic causes such as hypothyroidism or drug side effect
        - direct enquiry about suicidal ideas, delusions and hallucinations
        - review of related functional, interpersonal and social difficulties
        - evidence of self-neglect, psychosis or severe agitation
        - cultural factors

        - past psychiatric history including previous response to treatment
        - patient safety and risk to others
- associated diseases
    - dysthymia
        - chronic state of depression lasting more than two years which does not meet the criteria for major depression and is not the result of partially resolved major depression
    - eating disorders
    - substance misuse
    - other psychiatric disorders
    - some medical conditions have known associations with depression:
        - Parkinson's disease
        - chronic diseases such as diabetes and cardiac disease
        - cerebrovascular disease
        - endocrine disorders
        - cancer, especially pancreatic
        - autoimmune conditions
- investigations
    - used to exclude organic causes and not always required
    - may include:
        - bloods
            - glucose, U&Es, LFTs, TFTs, calcium, FBC, inflammatory markers, magnesium, HIV or syphilis serology, drug screening
        - imaging
            - MRI or CT where presentation or examination is atypical or there are features suggestive of intracranial lesion
- management
    - general measures
        - managing comorbidity
        - managing any safeguarding issues
        - assessing and mitigating suicide risk
        - appropriate monitoring/follow up
        - advising on sleep hygiene where relevant
    - management is generally through primary care
    - mild to moderate depression or persisting subthreshold depressive symptoms
        - consider watchful waiting with reassessment in around 2 weeks
        - low intensity psychosocial interventions (usually via referral to IAPT)
            - guided self-help based on CBT principles
            - physical activity programmes in facilitated group sessions
            - counselling or short-term psychodynamic psychotherapy
        - antidepressants are not recommended as the risk:benefit balance is poor
            - they may be considered if:
                - mild depression persists after other interventions or is associated with psychosocial or medical problems
                - mild depression complicates the care of physical health problems

        - a patient with a history of moderate to severe depression presents with mild depression
        - subthreshold depressive symptoms present for at least two years or persisting after other interventions
    - moderate to severe depression
        - antidepressant medication combined with high intensity psychological treatment
        - psychiatric review if there is suicidal ideation or plans, self or others at risk, psychosis, severe agitation or self-neglect
        - ECT may be considered for fast and short-term improvement of symptoms when all other treatment options have failed or the situation is life-threatening
    - antidepressants
        - SSRIs are first line
        - escitalopram is probably the best first choice
        - sertraline may be preferred when there are coexisting physical health problems, as there are fewer drug interactions
        - past treatments and the response to them should be considered
        - patients' fears of addiction and other concerns should be addressed before starting medication, and they should be informed about the delay in onset of effect (2-4 weeks) and time course of treatment (at least 6 months from remission of symptoms)
        - there is increased risk of bleeding with SSRIs
- complications
    - impaired quality of life and reduced productivity
    - social difficulties
    - increased mortality – death by suicide and increased mortality rate in comorbid conditions
    - exacerbation of pain and disability associated with physical conditions
- prognosis
    - average duration of a depressive episode is 6-8 months
        - spontaneous recovery is likely with mild depression
    - risk of recurrence is at least 50% after a first episode and higher after further episodes
    - prognosis is worse where there is prominent anxiety, underlying personality disorder or symptoms which are particularly severe
    - risk factors for increased risk of recurrence include:
        - ≥3 episodes of major depression
        - high prior frequency of recurrence
        - an episode in the previous 12 months
        - residual symptoms during continuation treatment
        - severe episodes (e.g. suicidality, psychotic features)
        - long previous episodes
        - relapse after drug discontinuation

MHC3 eating disorders

- bulimia nervosa
    - background

        - an eating disorder characterised by repeated episodes of uncontrolled eating (binges) followed by compensatory weight loss behaviours
        - features include:
            - excessive preoccupation with body weight and shape
            - undue emphasis on weight in self-evaluation
            - feeling of lack of control over eating
            - compensatory weight control mechanisms which can be:
                - self-induced vomiting
                - fasting
                - intensive exercise
                - abuse of medication such as laxatives, diuretics, thyroxine or amphetamines
        - there is crossover with binge eating disorder where there is bingeing behaviour without the compensatory purgeing
    - epidemiology
        - incidence is around 10-15 per 1000 females per year
        - lifetime prevalence in women is around 2%
        - occurs across all socio-economic groups
        - more common in western societies
        - occurs in ten times as many females as males
        - more common in adolescence and young adulthood
        - many do not seek treatment
    - risk factors
        - parental and childhood obesity
        - family dieting
        - family history of eating disorders
        - history of severe life stresses and possibly physical or sexual abuse
        - parental and premorbid psychiatric disorder or substance misuse
        - parental problems, such as high expectations, low care, overprotection and disruptive events in childhood such as parental death and alcohol dependency
        - early experiences of criticism regarding eating habits or body weight
        - perceived pressure to be thin (from cultural or family sources)
        - recreational pressure (models, jockeys, ballet dancers, athletes)
        - early menarche
        - premorbid characteristics such as perfectionism, anxiety, obsessional traits, low self-esteem, emotionally unstable personality disorder, difficulty in resolving conflict
    - history
        - regular binge eating with loss of control of eating during binges
            - to meet DSM-5 criteria it should occur on average at least once a week for 3 months
        - attempts to counteract the binges – e.g. vomiting, using laxatives, diuretics, dietary restriction, excessive exercise
        - BMI is maintained above 17.5
        - preoccupation with weight, body image and body shape; self-evaluation is unduly based on body weight and shape
        - preoccupation with food and diet

                - often rigid or ritualistic with deviations from a planned eating programme causing distress
                - the affected person starts to avoid eating with other people and becomes isolated
    - physical symptoms
        - bloating and fullness
        - lethargy
        - heartburn and reflux
        - abdominal pain
        - sore throat and dental problems due to vomiting
    - examination
        - usually normal
        - mainly aimed at excluding physical complications such as dehydration or dysrhythmias
        - examination must include height and weight with calculation of BMI and blood pressure
        - in severe cases signs may be present:
            - salivary glands (especially the parotid) may be swollen
            - there may be oedema if there has been laxative or diuretic abuse
            - Russell's sign may be present – calluses on the back of the hand caused by repeated abrasion against the teeth during inducement of vomiting
            - erosion of dental enamel due to repeated vomiting
    - investigations
        - usually normal apart from hypokalaemia
        - U&Es should be checked in view of frequent vomiting
    - management
        - patients need referral to an age-appropriate, community based specialist eating disorder service for assessment and management
        - the majority of patients can be treated as outpatients
        - NICE recommends an initial evidence-based bulimia-focused guided self-help programme
            - if ineffective or not an option, eating disorder focused CBT should be offered, usually 20 sessions over 20 weeks
        - for under 18s, NICE recommends bulimia-nervosa-focused family therapy
            - the alternative is individual eating-disorder-focused CBT
    - general medical aspects
        - assessment and monitoring of electrolytes in patients vomiting frequently or taking large amounts of laxatives
            - eliminating the behaviour responsible is usually sufficient to treat imbalances
            - regular dental reviews and dental hygiene (e.g. rinsing the mouth after vomiting)
            - slow reduction of laxatives
            - screening for osteoporosis
    - complications
        - haematemesis

        - metabolic complications
        - dental erosions
        - painless enlargement of the salivary glands
        - tetany
        - seizures
        - around 10-15% go on to develop anorexia
    - prognosis
        - up to 80% make a complete recovery with treatment
        - if recovery has not occurred within 5 years, they are more likely to progress to a chronic course
        - risk of death is significantly lower than with anorexia nervosa and estimated at around 0.4% due to the slight increase in suicide
- anorexia nervosa
    - background
        - maintenance of a low body weight as a result of preoccupation with weight, construed as either a fear of fatness or a pursuit of thinness
        - they believe they are fat and are terrified of becoming what is in fact a normal weight or shape
        - can cause widespread physical and psychological morbidity and can result in death
    - epidemiology
        - mean yearly incidence is around 0.4 per 1000 per year in females
        - 9 in 100 females will experience the condition at some point
        - affects women more than men
            - however men are likely to be underdiagnosed, misdiagnosed and under-referred
        - typical age of onset is early to mid adolescence
    - aetiology
        - likely to be multifactorial involving biological, psychological, developmental and sociocultural factors
        - the main risk factors are thought to be:
            - female gender
            - age
            - living in a western society
            - family history of eating disorder, depression or substance misuse
            - premorbid experiences including:
                - sexual abuse
                - dieting behaviour within family or personal experience
                - occupational or recreational pressure to be slim
                - onset of puberty
                - criticism or perceived criticism about weight or eating behaviour
            - personal characteristics
                - perfectionism
                - low self-esteem
                - obsessional traits
                - premorbid obesity

        - early menarche
        - difficulty with resolving conflict
        - anxiety
        - emotionally unstable personality disorder
- presentation
    - suspicion and diagnosis are based on history, suggestive clinical features and often concerns raised by a relative or friend
    - clinical features include:
        - refusal to maintain a normal body weight for age and height
        - weight below 85% of predicted (= BMI <17.5 in adults)
        - dieting or restrictive eating practices – change in eating behaviour may be reported by friends and family
        - rapid weight loss
        - having a dread of gaining weight
        - disturbance in the way weight or shape is experienced, resulting in over-estimation of size
        - denial of the problem
        - lack of desire for intervention, or resistance to it
        - social withdrawal, few interests
        - enhanced weight loss by over-exercise, diuretics, laxatives and self-induced vomiting
        - problems managing pre-existing chronic diseases which involve dietary control, such as diabetes or coeliac disease
    - other physical features include:
        - in women, amenorrhoea for 3 months or longer
        - gastrointestinal symptoms such as constipation, feeling of fullness after meals, dysphagia, abdominal pains
            - these may hinder diagnosis and treatment
        - symptoms such as fatigue, fainting, dizziness and intolerance of cold
        - delay in secondary sexual development if pre-puberty
- examination
    - should include:
        - height, weight and BMI
        - core temperature
        - peripheral examination – circulation, oedema
        - cardiovascular examination – pulse, BP, postural hypotension
        - testing of muscle power with the sit-squat-stand test
    - examination can be normal but possible findings include:
        - bradycardia
        - hypotension
        - peripheral oedema
        - gaunt face
        - lanugo hair
        - scanty pubic hair
        - acrocyanosis (red or purple hands or feet)
- investigations
    - ESR and TFTs to screen for other causes of weight loss

        - other tests depend on individual presentation
            - U&Es in patients with frequent vomiting, laxative or diuretic abuse
            - frequent bloods in patients with very low BMI or otherwise high risk
        - those below 18 years may need a DXA scan after a year of being underweight (earlier if fractures or bone pain) and after two years in adults
            - may need ongoing monitoring with DXA scans if remaining underweight (no more than yearly)
        - ECG may show bradycardia or prolonged QTc
    - management in the community
        - under 18s
            - anorexia-nervosa-focused family therapy
            - individual CBT or adolescent-focused psychotherapy
        - adults
            - individual eating-disorder-focused CBT
            - Maudsley Anorexia Nervosa Treatment for Adults (MANTRA)
            - specialist supportive clinical management (SSCM)
    - complications
        - hypokalaemia
            - common, and may cause fatal arrhythmias
        - hypotension
        - cardiac problems including arrhythmias, mitral valve prolapse, peripheral oedema, sudden death
        - anaemia and thrombocytopaenia
        - hypoglycaemia
        - osteoporosis
            - restoring the patient's weight is the best treatment
        - constipation
        - lack of growth in teenagers and lack of development of secondary sexual characteristics
        - infertility
        - infections
        - renal calculi
        - AKI or CKD
        - alcohol dependency in some cases
        - anxiety and mood disorders
        - social difficulties
    - prognosis
        - variable
        - highest mortality of all psychiatric conditions
            - due to medical complications and increased risk of suicide
        - around 50% make a full recovery, 33% improve, 20% have a chronic eating disorder
        - recovery rate is thought to be higher when onset is in adolescence (70-80% or more)
        - relapse is common
        - long term follow up studies put mortality rate as high as 18%

        - poor prognosis is predicted by a long duration of illness prior to presentation, the need for hospitalisation and onset in adulthood
        - there is a high risk of comorbid or subsequent psychiatric conditions such as anxiety disorders, OCD, depression, substance abuse
        - early detection may improve prognosis
- Management of Really Sick Patients with Anorexia Nervosa (MARSIPAN) key points
    - physical assessment
        - patients near to death often look well
        - BMI <13 or rapid weight loss (>1kg/week) are high risk
        - physical examination, including muscle power (SUSS test)
            - SUSS (sit up-squat-stand test)
                - sit up
                    - patient lies down flat on the floor and sits up, without, if possible, using their hands
                - squat-stand
                    - patient squats down and rises without, if possible, using their hands
                - scoring (for sit up and squat-stand tests separately)
                    - 0: unable
                    - 1: able using only hands to helps
                    - 2: able with noticeable difficulty
                    - 3. able with no difficulty
        - blood tests
            - especially electrolytes, glucose, phosphate, magnesium, LFTs, FBC
        - ECG
            - especially QTc interval, ST and T wave changes
        - high risk patients should not be discharged without specialist consultation
        - even mild hypokalaemia in eating disorders probably signifies low total body potassium and more severe hypokalaemia can recur after discharge with fatal results
    - nutritional issues
        - consult a medical expert in nutrition if possible
        - replace thiamine early and prescribe a vitamin and mineral supplement
        - avoid re-feeding syndrome by slow re-feeding and close monitoring in vulnerable patients
        - avoid underfeeding syndrome by frequent (12-hourly) reassessment and increasing calories as soon as safe
    - psychiatric issues
        - transfer to a specialist eating disorders unit if possible
        - regular liaison with a psychiatrist (eating disorders, liaison or community)
        - be aware of sabotaging behaviour such as falsifying weight, water drinking, exercising
        - use only experienced and trained nurses to observe
        - if staff inexperienced in management of anorexia nervosa are recruited, a concise management plan should be provided to follow
        - consider Mental Health Act section if a patient fails to improve
    - consult the MARSIPAN report
- MARSIPAN checklist (RCPsych)

- assessing
  - does the patient have anorexia nervosa?
    - yes
    - not sure and psychiatric review requested
  - are there significant risk factors?
    - BMI <13 (adults) or <70% BMI for age (under 18)
    - recent loss of ≥1kg for two consecutive weeks
    - little or no nutrition for >5 days
    - acute food refusal or <500kcal/day for >2 days in under 18s
    - pulse <40
    - BP low with postural dizziness
    - core temperature <35°
    - Na+ <130mmol/L
    - K+ <3.0mmol/L
    - raised transaminase
    - glucose <3mmol/L
    - raised urea or creatinine
    - ECG – e.g. bradycardia, QTc .450ms
  - is the patient consenting to treatment?
    - yes
    - no and assessment for compulsory detention requested
- refeeding
  - is intensive medical care needed?
    - yes
    - no and regular risk monitoring in place
  - increased risk of refeeding syndrome?
    - low initial electrolytes
    - low BMI
    - significant comorbidities (e.g. infection, cardiac failure, alcoholism, uncontrolled diabetes)
    - start at 5-10kcal/kg/day
    - monitor electrolytes twice daily and build up calories swiftly – avoid underfeeding
  - lower risk of refeeding syndrome?
    - start at 15-20kcal/kg/day and build up swiftly
    - avoid underfeeding syndrome
  - give all adults oral thiamine and Pabrinex
  - monitor
    - electrolytes
    - ECG
    - vital signs
    - BMI
- managing
  - are medical and psychiatric staff collaborating in care?
    - yes
    - no and psychiatric assessment awaited
  - are nurses trained in managing medical and psychiatric problems?

                - yes
                - no and appropriately skilled staff requested/training in place
            - are there behaviours that increase risk? (mobilise psychiatric team to advise on management)
                - purging behaviours
                - falsifying weight
                - disposing of feed
                - exercising
                - self-harm, suicidality
                - family distress/anxiety
                - safeguarding concerns

MHC4 personality disorders

- background
    - ICD-10 (WHO) definition of personality disorder
        - a severe disturbance in the characterological condition and behavioural tendencies of the individual, usually involving several areas of the personality and nearly always associated with considerable personal and social disruption
    - DSM-IV (American Psychiatric Association) definition
        - an enduring pattern of inner experience and behaviour that differs markedly from the expectations of the individual's culture, is pervasive and inflexible, has an onset in adolescence or early adulthood, is stable over time and leads to distress or impairment
    - aetiology is uncertain
    - affects 4-11% of the UK population and 60-70% of the prison population
    - factors in childhood postulated to be linked to personality disorders:
        - sexual abuse
        - physical abuse
        - emotional abuse
        - neglect
        - being bullied
    - emotional or behavioural factors that may play a part include:
        - truanting
        - bullying others
        - being expelled/suspended
        - running away from home
        - deliberate self-harm
        - prolonged periods of misery
    - there is a growing evidence base suggesting a role for genetic factors
    - people with personality disorders are at increased risk for many psychiatric disorders
- classification (ICD-10)
    - paranoid personality disorder
    - schizoid personality disorder
    - dissocial personality disorder
    - emotionally unstable personality disorder

        - impulsive type
        - borderline type
    - histrionic personality disorder
    - anankastic personality disorder
    - anxious (avoidant) personality disorder
    - dependent personality disorder
    - other specific personality disorders including:
        - eccentric
        - 'Haltlose' type
        - immature
        - narcissistic
        - passive-aggressive
        - psychoneurotic
    - unspecified personality disorder
- presentation (ICD-10)
    - markedly disharmonious attitudes and behaviour, involving usually several areas of functioning (e.g. affectivity, arousal, impulse control, relationships with others)
    - the abnormal behaviour pattern is persistent, lasts for a long time and is not limited to episodes of mental illness
    - the abnormal behaviour pattern is widespread and obviously maladaptive to a broad range of personal and social situations
    - the above manifestations always appear during childhood or adolescence and continue into adulthood
    - the disorder leads to considerable personal distress but this may only become apparent at a later stage
    - the disorder is usually, but not always, associated with significant difficulties with work and social relationships; clinically significant distress or impairment must occur in all settings and not be limited to one area only
    - common presenting features:
        - paranoid
            - displaying pervasive distrust and suspicion
            - common beliefs include:
                - others are exploiting or deceiving them
                - friends and associates are untrustworthy
                - information confided to others will be used maliciously
                - there is hidden meaning in remarks or events other perceive as benign
                - the spouse or partner is unfaithful
        - schizoid
            - withdrawal from affectional, social and other contacts; the person is isolated and has a limited capacity to experience pleasure and express feelings
            - may speak with odd or idiosyncratic use of language
        - dissocial
            - a tendency to act outside social norms, a disregard for the feelings of others and an inability to modify behaviour in response to

adverse events (e.g. punishment); may have a low threshold for violence and a tendency to blame others
                - may display a hostile attitude
            - emotionally unstable
                - people are impulsive and unpredictable and may act without appreciating the consequences; may exhibit outbursts of emotion and quarrelsome behaviour; relationships tend to be unstable and there may be suicidal gestures and attempts
            - histrionic
                - characterised by shallow and labile affectivity and theatricality; lack of consideration for others and a tendency for egocentricity; often crave excitement and attention
                - may display 'la belle indifférence' – a seemingly indifferent detachment whilst describing dramatic physical symptoms
            - anankastic
                - characterised by feelings of doubt, perfectionism and excessive conscientiousness; compulsion to check and preoccupation with details; tend to be stubborn, cautious and rigid; may have intrusive insistent and unwelcome thoughts or impulses that do not meet the criteria for OCD
            - anxious (avoidant)
                - feelings of tension and apprehension, insecurity and inferiority; people yearn to be liked and accepted and are sensitive to rejection; tendency to exaggerate potential dangers and risks, leading to an avoidance of everyday activities
            - dependent
                - characterised by a reliance on others to take decisions and a fear of abandonment; excessive reliance on authority figures and difficulty in acting independently; can affect the capacity to deal with the intellectual and emotional demands of daily life
- investigations
    - Minnesota Multiphasic Personality Inventory (MMPI)
    - Eysenck Personality Inventory and the Personality Diagnostic Questionnaire
    - structed psychometric assessment
- management
    - psychotherapy
        - psychodynamic psychotherapy
        - CBT
        - interpersonal therapy
        - group psychotherapy
        - dialectical behavioural therapy
    - pharmacotherapy
        - may be suitable for some disorders
            - mood stabilisers and antipsychotics for specific symptoms in borderline personality disorder
            - antipsychotics or benzodiazepines short term for crisis periods
- crisis management

    - consult patient's crisis plan
    - assess problem and risk
        - maintain a calm and non-threatening attitude
        - try to understand the crisis from the person's point of view
        - explore the person's reason for distress
        - use empathetic open questioning, including validating statements, to identify the onset and the course of the current problems
        - seek to stimulate reflection about solutions
        - avoid minimising the person's stated reasons for the crisis
        - wait for full clarification of the problems before offering solutions
        - explore other options before considering admission to a crisis unit or inpatient admission
        - offer appropriate follow-up within a timeframe agreed with the person
        - assess risk to self and others
        - ask about previous episodes and effective management strategies used in the past
        - help to manage their anxiety by enhancing coping skills and helping them to focus on the current problems
        - encourage them to identify manageable changes that will enable them to deal with the current problems
        - make sure they have follow up at an appropriate time (e.g. with GP)
    - referral to crisis team:
        - levels of distress and/or risk of harm to self or others is increasing
        - levels of distress and/or risk of harm to self or others has not subsided despite attempts to reduce anxiety and improve coping skills
        - patient requests further help from specialist services
- complications
    - suicide
    - substance abuse
    - accidents and injuries
    - depression
    - homicide
- emotionally unstable personality disorder (borderline personality disorder)
    - background
        - unknown cause, likely an interaction between adverse life events and genetic factors
        - pattern of sometimes rapid fluctuation from periods of confidence to despair, with fear of abandonment and rejection
        - strong tendency towards suicidal thinking and self-harm
        - may have transient psychotic symptoms
        - patients are at risk of suicide
        - prevalence in the general population is 1%
        - more common in females
    - diagnosis
        - the general criteria of personality disorder must be met
        - impulsive type
            - emotional instability and lack of impulse control

                - outbursts of violence or threatening behaviour are common, particularly in response to criticism
            - borderline type
                - several of the characteristics of emotional instability are present
                - patient's own self-image, aims and internal preferences (including sexual) are often unclear or disturbed
                - chronic feelings of emptiness
                - liability to become involved in intense and unstable relationships which may cause repeated emotional crises and may be associated with excessive efforts to avoid abandonment and a series of suicidal threats or self-harm
    - presentation
        - relationship difficulties
        - recurrent self-harm
        - threats of suicide
        - depression
        - bouts of anger
        - impulsivity
        - social difficulties
        - transient psychotic symptoms
    - investigation
        - may include toxicology screen
        - HIV/STI testing may be appropriate because of poor impulse control and disregard of risk
        - psychological testing as per NICE:
            - Diagnostic Interview for DSM-IV Personality Disorders (DIPD-IV)
            - Structured Clinical Interview for DSM-IV Personality Disorders (SCID-II)
            - Structured Interview for DSM-IV Personality (SIDP-IV)
            - International Personality Disorder Examination (IPDE)
            - Personality Assessment Schedule (PAS)
            - Standardised Assessment of Personality (SAP)
    - associated diseases
        - anxiety
        - alcohol misuse
        - drug misuse
        - depression
        - recurrent self-harm
        - eating disorders
        - post-traumatic stress disorder
        - physical conditions:
            - arteriosclerosis
            - hypertension
            - hepatic disease
            - cardiovascular disease
            - gastrointestinal disease
            - arthritis

                - sexually transmitted infections
        - management
            - patients should have a crisis plan which:
                - identifies potential tiggers that could lead to a crisis
                - specifies self-management strategies likely to be effective
                - establishes how to access services
            - psychotherapy
            - drug treatment
                - rarely indicated but may be used for specific symptoms
- dissocial personality disorder
    - background
        - patients have traits of impulsivity, high negative emotionality, low conscientiousness and associated behaviours, including irresponsible and exploitative behaviour, recklessness and deceitfulness
        - they have often grown up with parental conflict and harsh inconsistent parenting
        - childhoods typically features parental inadequacies and often transfer of care to outside agencies
        - there is a high incidence of truancy, delinquency and substance misuse, resulting in increased rates of unemployment, problems with housing and difficulties with relationships
        - many have a criminal conviction, are imprisoned or die early as a result of reckless behaviour
        - prevalence is likely 0-1%, and higher in men
    - presentation
        - features include:
            - unstable interpersonal relationships
            - disregard for the consequences of their behaviour
            - a failure to learn from experience
            - egocentricity
            - a disregard for the feelings of others
            - a wide range of interpersonal and social disturbance
            - comorbid depression and anxiety
            - comorbid alcohol and drug misuse
    - diagnostic criteria
        - general criteria of personality disorder must be met
        - at least three of the following:
            - callous unconcern for the feelings of others
            - gross and persistent attitude of irresponsibility and disregard for social norms, rules and obligations
            - incapacity to maintain enduring relationships, although having no difficulty to establish them
            - very low tolerance to frustration and a low threshold for discharge of aggression, including violence
            - incapacity to experience guilt, or to profit from adverse experience. particularly punishment

                - marked proneness to blame others, or to offer plausible rationalisations for the behaviour bringing the subject into conflict with society
        - management
            - patients can create very difficult and frightening problems for staff in healthcare
            - it is important to identify patients at risk of violent behaviour
                - current or previous violence, including severity, circumstances, precipitants and victims
                - presence of comorbid mental disorders and/or substance misuse
                - current life stressors, relationships and life events
                - additional information from written records or families and carers if possible as the person may not always be reliable
                - use of chaperones, panic buttons and other measures should be considered
            - drug treatment
                - only to treat specific symptoms
            - psychological therapies
                - tries to improve perceptions of and responses to social and environmental stressors

MHC5 acute psychosis including bipolar, schizophrenia

- definitions
    - psychosis
        - a group of mental disorders that feature loss of contact with reality
    - delusion
        - an irrationally held belief that cannot be altered by rational argument
    - delirium
        - acute disorder of mental processes accompanying organic disease
    - hallucination
        - a false perception in the absence of external stimuli
    - dementia
        - an acquired global impairment of intellect, memory and personality without impairment of consciousness
- pathophysiology
    - likely to in part be due to genetic, biochemical and structural abnormalities
    - potential organic causes of altered mental state should be ruled out
        - structural abnormalities
            - space occupying lesions
        - biochemical changes
            - electrolyte imbalance
        - physiological changes
            - hypotension
        - multi-factorial conditions
            - sepsis
- assessment and risk stratification
    - factors to consider:

        - personal privacy issues
            - avoid giving out addresses or personal details of staff
            - consider whether addresses are obtainable from other sources (e.g. GMC database of registered professionals)
        - personal appearance
            - avoid loose clothing that could be used to harm
            - avoid wearing provocative or offensive items
        - personal behaviour
            - maintain professional behaviour
            - be positive and courteous
        - isolation
            - check the known risks of patients prior to assessment
            - ensure other staff are aware of your location and that you know how to raise the alarm should you feel threatened
        - emergency situations
            - leave the room if possible
            - raise the alarm to alert other staff members
        - recommendations for safety during assessment
            - no ligature points
            - nothing that can be used as a missile
            - two doors that open both ways
            - chaperone present during an interview and regular checks provided by other staff, e.g. every five minutes
            - psychiatric assessment should be available within the emergency department within one hour
            - mental health nurses should be employed within the emergency department
            - lorazepam is recommended as first line for rapid tranquilisation
    - confidentiality
        - patients should be able to disclose information in a safe and private environment
        - relatives, friends and third parties (e.g. police) should not be informed of what occurs during the consultation
        - there are specific instances where confidentiality can be breached, as per GMC guidance
            - the patient's consent should be sought before breaching confidentiality, and if they refuse it should be discussed with the Trust's legal department and your defence union
        - examples of where confidentiality can be breached include disclosures:
            - required by law
                - e.g. notification of a communicable disease
            - relating to the courts or litigation
                - e.g. specific requests from a judge for relevant information
            - relating to statutory regulatory bodies
                - e.g. DVLA where a patient poses a threat to the public
            - in the public interest

        - e.g. the patient has disclosed involvement in serious criminal activity such as terrorism
    - to protect the patient or others
        - e.g. patient expresses homicidal intent towards a specific person
- triage and assessment of risk of self-harm
    - the mental health triage scale can be used at the initial assessment of the patient (developed for use with the NICE guidelines on self-harm)
        - can be helpful to assess risk of further self-harm, risk to others and need for higher level of supervision prior to full assessment
    - various suicide and self-harm risk assessment tools are available, including:
        - Pierce suicide risk assessment tool
        - Edinburgh risk of repetition tool
        - Beck suicide intent tool
            - they have a low accuracy for prediction of further self-harm but can prompt referral for assessment
- UK Mental Health Triage Scale typical presentations
    - triage code A (emergency)
        - current actions endangering self or others
        - overdose/suicide attempt/violence or aggression
        - possession of a weapon
    - triage code B (very high risk of imminent harm to self or others)
        - acute suicidal ideation or risk of harm to others with clear plan or means
        - ongoing history of self-harm or aggression with intent
        - very high risk behaviour associated with perceptual or thought disturbance, delirium, dementia or impaired impulse control
    - triage code C (high risk of harm to self or others and/or high distress, especially in absence of capable supports
        - suicidal ideation with no plan or ongoing history of suicidal ideas with possible intent
        - rapidly increasing symptoms of psychosis and/or severe mood disorder
        - high risk behaviour associated with perceptual or thought disturbance, delirium, dementia or impaired impulse control
        - overt/unprovoked aggression in care home or hospital ward setting
        - wandering at night (community)
        - vulnerable, isolation or abuse
    - triage code D (moderate risk of harm and/or significant distress)
        - significant patient/carer distress associated with severe mental illness (but not suicidal)
        - absent insight/early symptoms of psychosis
        - resistive aggression/obstructed care delivery
        - wandering (hospital) or during the day (community)
        - isolation/failing carer or known situation requiring priority intervention or assessment
    - triage category E (low risk of harm in short term or moderate risk with good support/stabilising factors)
        - requires specialist mental health assessment but is stable and low risk of harm during waiting period

        - other services able to manage the patient until mental health service assessment
        - known service user requiring non-urgent review, adjustment of treatment or follow-up
        - referral for diagnosis
        - requests for capacity assessment, service access for dementia or service review/carer support
    - triage category F (referral not requiring face to face response from Mental Health)
        - other services outside Mental Health more appropriate to current situation or need
    - triage category G (advice, consultation, information)
        - patient or carer requiring advice or information
        - service provider providing information (collateral)
        - initial notification pending further information or detail
- examination
    - assess whether the patient is capable of giving accurate information
    - mental state assessment
        - Folstein mini-mental state assessment
            - has 30 questions and can be time consuming
        - the abbreviated mental test
            - more appropriate to the ED
            - a score of 6 or less indicates cognitive impairment
            - questions
                - age
                - date of birth
                - what year it is
                - what time it is
                - what address/place we are in
                - register a 3 line address and recall at the end
                - who the king or queen is
                - the year of World War 1
                - counting backwards from 20-1
                - identify two people (names/jobs)
- psychiatric history (components to include)
    - identifiers
        - name
        - age
        - occupation
        - location of consultation
        - Mental Health Act status
    - presenting complaint
        - brief description, ideally in the patient's own words, of why they are attending the department
    - history of presenting complaint
        - time of onset
        - course
        - progression

        - associations
        - severity
        - predisposing factors
    - past psychiatric history, past medical history, past surgical history
        - all previous admissions to psychiatric units
        - MHA category for previous admissions
        - current and previous psychiatric contacts
    - drug history
        - current medications, dosages, routes and frequencies
        - known allergies, reactions and intolerances
    - social history
        - employment status
        - living arrangements
        - smoking, drug and alcohol history
        - normal functional capacity (e.g. mobility and independence)
    - family history
        - any conditions in the immediate family
    - personal history
        - childhood development
        - family tree
        - parental issues
        - schooling
        - employment
        - relationships
        - children
        - social circumstances
        - financial issues
        - forensic issues
- key components of mental state examination
    - appearance
        - dress unkempt
        - self-harm
        - physical disorder
        - inappropriate dress
        - previous deliberate self-harm
    - behaviour
        - co-operation
        - aggression
        - disinhibition
        - distress
        - preoccupations
    - speech
        - amount
        - spontaneity
        - volume
        - flow
    - mood and affect
        - subjective versus objective

        - posture
        - facial expression
    - suicidality
        - plans and intent
    - thought form
        - tangentiality
        - circumstantiality
        - associations
        - neologisms
        - flight
    - thought content
        - worries
        - obsessions
        - delusions
    - abnormal perception
        - hallucination and sensory modalities
    - cognition
        - orientation
        - attention
        - memory
        - intelligence
    - insight
        - patient's understanding and expectations of their condition
- consideration of organic causes
    - signs and symptoms suggestive of an organic cause:
        - history of substance misuse
        - over 35 years at first presentation
        - fluctuating behaviour
        - predominantly visual hallucinations
        - lethargy
        - abnormal vital signs
        - poor cognitive function
    - examples of organic causes:
        - traumatic
            - head injury, acute and long-term
        - infective
            - HIV
            - encephalitis
            - syphilis
            - malaria
            - meningitis
            - cerebral toxoplasmosis
        - neurological
            - Parkinson's disease
            - CVA
            - epilepsy
            - Huntington's disease

        - cardiac
            - mitral valve disease
        - autoimmune
            - SLE
        - neoplasia
            - frontal lobe
            - temporal lobe
            - paraneoplastic problems (e.g. hypercalcaemia)
        - blood/bone
            - haemolytic anaemias
            - thrombotic thrombocytopenic purpura
        - endocrine
            - adrenocortical diseases
            - thyroid disease
            - phaeochromocytoma
        - degenerative
            - dementia
            - visual hallucinations in partially sighted
        - drugs
            - alcohol
            - illicit drugs
            - steroids
        - idiopathic
            - genetic disorders (e.g. Huntington's, storage disorders, Wilson's)
        - obstetric/gynaecological
            - pre-eclampsia
            - post-partum
- indications for admission for patients with a psychiatric diagnosis
    - first episode of psychosis
    - suicidal or homicidal
    - gross debilitation from illness
    - lack of capacity to consent to or comply with treatment
    - inadequate psychosocial support in the community
- bipolar disorder
    - background
        - a chronic episodic illness associated with behavioural disturbances
        - bipolar I
            - presents with manic episodes, most commonly interspersed with major depressive episodes
            - manic episodes are severe and result in impaired functioning and frequent hospital admissions
        - bipolar II
            - patients do not meet the criteria for full mania and are described as hypomanic
            - hypomania has no psychotic symptoms and results in less associated dysfunction
            - often interspersed with depressive episodes

- epidemiology
  - lifelong prevalence rate of around 2.4%
  - similar rates in males and females
  - relatives are 5-10 times more likely to have the disorder themselves
- presentation
  - manic phase
    - characterised by elevated mood and increase in quantity and speed of physical and mental activity
    - some patients may be excessively happy, whilst others may be irritable and easily angered
    - possible features include
      - grandiose ideas
      - pressure of speech
      - excessive amounts of energy
      - racing thoughts and flight of ideas
      - overactivity
      - needing little sleep, or an altered sleep pattern
      - easily distracted – starting many activities and leaving them unfinished
      - bright clothes or unkempt
      - increased appetite
      - sexual disinhibition
      - recklessness with money
    - severe cases may have grandiose delusions, auditory hallucinations, delusions of persecution and lack of insight
  - hypomanic phase
    - lesser degree of mania with persistent mild elevation of mood and increased activity and energy but without hallucinations or delusions
    - no significant effect on functional ability
  - depressive phase
    - low mood and reduced energy
    - no joy in daily activities and negative thoughts
    - they lack facial expressions and have poor eye contact and may be tearful or unkempt
    - low mood is worse in the mornings and disproportionate to the circumstances
    - there may be feelings of despair, low self-esteem and guilt for which there may be no clear reason
    - there may be reduced appetite, weight loss, altered sleep pattern, loss of libido
    - in severe cases there may be delusions of persecution or illness or impending death
    - patients may become unwell through self-neglect
- diagnosis
  - at least two episodes in which a person's mood and activity levels are significantly disturbed (one of which must be mania or hypomania)
    - three of the following confirm mania:

        - grandiosity/inflated self-esteem
        - decreased need for sleep
        - pressured speech
        - flight of ideas
        - distractibility
        - psychomotor agitation
        - excessive involvement in pleasurable activities without thought
- clinical course
    - frequency and duration of episodes are variable
    - symptoms may vary from day to day and also within the day
    - patients may lead a normal lifestyle between episodes
    - 10-20% have rapid cycling (four or more cycles a year)
- reasons to be seen by the mental health team include:
    - severe depression
    - patient deemed to be a danger to themselves or others
    - poor or partial response to treatment
    - significant decline in function
    - depression associated with a period of overactivity or disinhibited behaviour lasting more than four days
    - poor adherence to treatment
    - intolerance to medication
    - comorbid misuse of alcohol or drugs
    - planning to stop medication after a period of stability
    - bipolar disease in pregnancy or if a woman is planning pregnancy
- management
    - non-pharmacological
        - education regarding diagnosis, treatment and side effects
        - good communication
        - self-help groups
        - support groups
        - self-monitoring of symptoms, side effects and triggers
        - coping strategies
        - psychological therapy
        - encouragement of engagement in calming activities
        - telephone support
    - pharmacological
        - manic episodes require urgent control and patients may be violent
        - patients should ideally be convinced to have oral therapy voluntarily
            - IM response is difficult to determine as absorption varies
            - therapy can be given by coercion under Common Law if not doing so would cause harm to the patient or others
            - rapid tranquilisation may be required
                - this can be achieved with antipsychotics, benzodiazepines or antihistamines orally, IM or rarely IV

                - first line treatments are antipsychotics, e.g. haloperidol, olanzapine, quetiapine, risperidone
                - if these do not work and the patient agrees to regular monitoring, lithium can be added
                - otherwise valproate can be added (not routinely in females of childbearing potential)
        - prognosis
            - requires lifelong treatment
            - a person with bipolar disorder experiences around 10 episodes in a lifetime
            - poor prognosis is associated with:
                - substance dependency
                - psychotic features
                - depression symptoms
                - interepisode depression
                - male gender
- schizophrenia
    - background
        - most common form of psychosis
        - can be chronic or relapsing and remitting
        - men are at higher risk of psychotic disorders, as are ethnic minorities
        - most commonly starts in adolescence or early 20s but can start at any time
    - aetiology
        - multiple factors are involved – genetic, environmental, social
        - cannabis use can increase risk
        - risk factors include:
            - family history
            - intrauterine and perinatal complications (e.g. premature birth, low birth weight)
            - intrauterine infection, especially viral
            - abnormal early cognitive/neuromuscular development
            - social isolation, migrants
            - abnormal family interactions (e.g. hostile or over-critical parents)
    - presentation
        - acute symptoms
            - hallmark symptoms are:
                - delusions
                - hallucinations
                - thought disorder
                - lack of insight
        - ‘first rank’ or ‘positive’ symptoms are rare in other psychotic illness; only one of the following symptoms is strongly predictive of the diagnosis:
            - lack of insight
            - auditory hallucinations, especially the echoing of thoughts or a third person ‘commentary’ on one’s actions
            - thought insertion, removal or interruption – delusions about external control of thought

                - thought broadcasting – the delusion that others can hear one's thoughts
                - delusional perceptions (i.e. abnormal significance for a normal event) - -e.g. 'the rainbow came out and I knew I was the son of God'
                - external control of emotions
                - somatic passivity – thoughts, sensations and actions are under external control
            - chronic symptoms ('negative symptoms')
                - underactivity – also affects speech
                - low motivation
                - social withdrawal
                - emotional flattening
                - self-neglect
    - signs
        - appearance and behaviour
            - withdrawal, suspicion or (rarely) stereotypical behaviours (repetition of purposeless movements) and mannerisms (e.g. saluting)
        - speech
            - interruptions to the flow of thought (thought blocking)
            - loosening of associations/loss of normal thought structure (knight's move thinking)
        - mood/affect
            - flattened, incongruous or 'odd'
        - abnormal beliefs
            - delusional perceptions, delusions concerning thought control or broadcasting, passivity experiences
        - abnormal experiences
            - hallucinations, especially auditory
        - cognition
            - attention, concentration, orientation and memory should be assessed (significant impairment suggests delirium or severe dementia)
    - associated conditions
        - depression
        - anxiety
        - post-traumatic stress disorder
        - personality disorder
        - substance misuse
        - obesity
        - diabetes
        - infections
        - cardiovascular disease
        - continuing disability
    - management
        - requires multidisciplinary support with joint primary and secondary care management

        - psychological support
            - includes information, support groups, family therapy, CBT
        - drugs
            - newer atypical antipsychotics are first line (e.g. risperidone, olanzapine)
                - depots can be considered
                - the main side effect is weight gain
                - they can rarely cause bone marrow depression
    - prognosis
        - 80% of people show some response to treatment in the first year
        - around 15% of people have persistent psychotic symptoms unresponsive to treatment two years after the acute episode
        - relative risk for suicide is increased 12-fold
        - all cause mortality is almost twice that of the general population
        - good prognostic factors include:
            - absence of family history
            - good premorbid function – stable personality and relationships
            - clear precipitant
            - acute onset
            - mood disturbance
            - prompt treatment
            - maintenance of initiative, motivation
        - poor prognostic factors include:
            - longer duration of untreated psychosis
            - early or insidious onset of schizophrenia
            - male sex
            - negative symptoms
            - family history of schizophrenia
            - low IQ, low socio-economic status or social isolation
            - significant psychiatric history
            - continued substance misuse

MHC6 somatic symptom disorders

- background
    - a chronic condition in which there are numerous physical complaints
    - ICD-10 definition:
        - multiple, recurrent and frequently changing physical symptoms usually present for several years (at least two years) before the patient is referred to a psychiatrist
    - DSM-IV definition allows patients who have underlying physical complaints to be included in the diagnosis
    - more common in patients with irritable bowel disorder and chronic pain
    - associated with PTSD and antisocial personality disorder
    - the somatising patient seems to seek the sick role, which offers relief from stressful or impossible interpersonal expectations (primary gain)
        - in most societies this results in attention, caring and sometimes monetary reward (secondary gain)

        - it is not malingering, as the patient is not aware of the process through which the symptoms arise, cannot will them away and genuinely experiences the symptoms
    - usually begins before the age of 30 and is more common in women
- presenting features
    - DSM-5
        - criteria rely less on strict patterns of symptoms and more on the degree to which a patient's thoughts, feelings and behaviours about their symptoms are disproportionate and excessive
        - symptoms are generally severe enough to affect work and relationships and for the patient to consult a doctor and take medication
        - stress often worsens the symptoms
    - symptoms include:
        - cardiac
            - shortness of breath
            - palpitations
            - chest pain
        - gastrointestinal
            - vomiting
            - abdominal pain
            - difficulty with swallowing
            - nausea
            - bloating
            - diarrhoea
        - musculoskeletal
            - pain in the legs or arms
            - back pain
            - joint pain
        - neurological
            - headaches
            - dizziness
            - amnesia
            - vision changes
            - paralysis or muscle weakness
        - urogenital
            - pain during urination
            - low libido
            - dyspareunia
            - impotence
            - dysmenorrhoea, irregular menstruation, menorrhagia
- diagnosis
    - physical examination and tests as per presenting complaint to rule out physical causes
    - psychological evaluation
    - somatisation is often a diagnosis of exclusion, but it is more effective to pursue a positive diagnosis when the patient presents with typical features:
        - there may be multiple symptoms, often occurring in different organ systems

        - symptoms that are vague or that exceed objective findings
        - chronic course
        - presence of a psychiatric disorder
        - history of extensive diagnostic testing
        - rejection of previous physicians
    - the clinician's emotional response to the patient can be helpful
        - a feeling of frustration or anger at the number and complexity of symptoms and the time required to evaluate them in an apparently well person
        - a sense of being overwhelmed by a patient who has had numerous evaluations by other physicians
- management
    - explanations of symptoms are often at odds with the patient's thinking and explanations should be tailored to be empowering, e.g.:
        - 'the results of my examination and of the tests we conducted show that you do not have a life-threatening illness. However, you do have a serious and impairing medical condition, which is often seen but not completely understood. Although no treatment is available that can cure it completely, there are a number of interventions that can help you deal with the symptoms better than you have so far.'
    - underlying mood disorders may be helped by antidepressants
    - the BATHE technique provides a framework for exploration of psychosocial stressors in less than 5 minutes:
        - background: 'what is going on in your life?'
        - affect: 'how do you feel about it?'
        - trouble: 'what troubles you the most about that situation?'
        - handle: 'what helps you handle that?'
        - empathy: ' this is a tough situation to be in; your reaction makes sense to me.'
    - avoid unrealistic goals
        - in severe cases, symptoms are unlikely to resolve completely, so the goal should not be to relieve the patient's illness (which would lead to frustration and possibly further investigations)
        - attempts to 'take away' the symptom may result in it being replaced with a different one as a result of the need to be sick
        - the goal should be to help the patient cope with the symptoms and keep the patient out of hospital
    - physical exercise and the enjoyment of private time are important
    - psychotherapy can be helpful, including CBT-type approaches and mindfulness
    - it is important to remember that somatising patients can still develop serious organic conditions, and prevention and screening should be a part of their plan
- complications
    - can result from invasive testing and multiple evaluations
    - dependency on analgesics or sedatives may develop
    - a poor relationship with the healthcare provider or evaluation by many providers can worsen the situation

MHC7 stress disorders

- stress

- definitions vary – in essence it is the autonomic 'alarm' response to perceived threat in the environment involving heightened arousal, adrenaline production facilitating short term 'fight or flight' resistance, followed by physical and mental exhaustion
    - commonly understood as a mismatch between external demands and the individual's ability to cope
    - many attribute physical illness to it
- individuals vary in their resilience to stress
    - some actively search for and thrive in high stress environments
    - others shun it and experience unhappiness, absenteeism and physical illness when stressed
    - life events such as bereavement, divorce and unemployment are all important stressors but normal adjustment reactions should not be medicalised

- acute stress reaction
    - background
        - a psychological condition that can develop after exposure and as a response to a stressful event
        - there is no 'acute stress disorder' in ICD-10 and acute stress reaction is defined as:
            - a transient disorder that develops in an individual without any other apparent mental disorder in response to exceptional physical and mental stress and that usually subsides within hours or days. Individual coping capacity and vulnerability play a role in the occurrence and severity of acute stress reactions
        - one of the key features is that it is believed to arise as a direct consequence of an exceptionally stressful life event and would not have occurred without this
    - traumatic events and risk factors
        - events may vary hugely and are specific to the individual
        - the precipitating event usually is or is perceived as life-threatening
        - can also result from sexual assault or ongoing trauma such as domestic violence
        - witnesses to events can also be at risk of acute stress reactions
        - refugees and asylum seekers at are much greater risk of acute stress reactions and PTSD
        - first responders are also at greater risk, and the possible self-selection for inherent resilience cannot be assumed to be protective
    - diagnosis
        - pattern of symptoms (ICD-10)
            - initial state of 'daze' with some constriction of the field of consciousness and narrowing of attention, inability to comprehend stimuli and disorientation
            - may be followed by further withdrawal from the surrounding situation or by agitation and over-activity (flight reaction or fugue)
            - autonomic signs of panic (tachycardia, sweating, flushing) are commonly present

- symptoms normally occur within minutes and disappear within 2-3 days (often within hours)
- partial or complete amnesia for the episode may be present
- if symptoms persist, a change in diagnosis should be considered

- further symptoms:
  - symptoms of intrusion, avoidance and hyperarousal
    - intrusion
      - often described as re-experiencing
      - spontaneous memories of the traumatic event or recurrent dreams and/or flashbacks
      - typically intense and cause psychological distress
    - avoidance
      - the person will try to avoid expressing thoughts or feelings which will trigger reminders of the event
    - hyperarousal
      - could be expressed through reckless or aggressive behaviour
      - can be self-destructive
      - there may be sleep disturbance and people can be hypervigilant (e.g. easily startled)
    - mood-related
      - may involve negative thoughts, moods or feelings
      - may feel estranged from others, blame themselves or have reduced enjoyment and interest in activities
    - these lead to impairment of social functioning and daily life
  - acute traumatic stress is generally limited to the first month and patients should be assessed for PTSD if it lasts longer

- management
  - no treatment may be required as symptoms may abate within hours or days
  - for those with ongoing symptoms, options include:
    - trauma-focused CBD
      - usually involves exposure challenge and/or direct challenge of negative trauma-related thoughts
    - eye movement desensitisation and reprocessing (EMDR)
      - aims to reduce stress in the shortest period of time
      - assumes that unprocessed memories are the cause of negative thoughts and feelings
  - medication
    - benzodiazepines should not be offered (WHO). neither should hypnotics (advice on relaxation techniques and sleep hygiene should be given)
    - beta-blockers can help reduce physical symptoms caused by the release of stress hormones
      - they can be taken as required and do not cause drowsiness, affect performance or become addictive
- prognosis
  - in most cases symptoms will subside in a few days, and less than a month

- post traumatic stress disorder
    - NICE definition
        - develops following a stressful event or situation of an exceptionally threatening or catastrophic nature, which is likely to cause pervasive stress in almost everyone
    - background
        - first recognised in WWI in men subjected to prolonged and intensive bombardment including gas attacks – was called 'shell shock'
        - recognised by DSM-III in 1980 after the Vietnam War
        - neurobiology likely involves the autonomic system and the hypothalamic-pituitary-adrenal axis, with noradrenaline being the main neurotransmitter involved
    - risk factors
        - precipitating event is or is perceived as being life-threatening
        - can be single or ongoing event
        - refugees and asylum seekers are at much greater risk of PTSD
        - first responders are more likely to be exposed to traumatic events
        - in the military, risk factors for PTSD include:
            - duration of combat exposure
            - low morale
            - poor social support
            - lower rank
            - unmarried
            - low educational achievement
            - history of childhood adversity
        - history of previous psychiatric disorders increases the risk of PTSD
    - history
        - recognition can be a challenge
        - starts soon after the event but may present later in 85%
        - symptoms fall into three categories:
            - re-experiencing
                - flashbacks where it seems as if the event were happening again
                - nightmares, which are common and repetitive
                - distressing images or other sensory impressions from the event which intrude during the waking day
                - reminders of the traumatic event provoke distress
            - avoidance or rumination
                - avoidance of reminders of the trauma, such as situations, people or circumstances associated with the event or resembling it
                - they may try to suppress memories or avoid thinking about the worst aspects
                - others ruminate excessively and prevent themselves from coming to terms with the experience, including on why it happened, whether it could have been prevented and whether revenge is possible

- hyperarousal or emotional numbing
    - may manifest as:
        - hypervigilance for threat
        - exaggerated startle responses
        - irritability
        - difficulty concentrating
        - sleep problems
        - difficulty experiencing emotions
        - feeling of detachment from others
        - giving up previously significant activities
        - amnesia for salient aspects of the trauma
    - children may have different presentation, including joylessly re-enacting the situation, having frightening dreams/sleep disturbance or behavioural changes
- screening
    - screening is of value
        - only those at high risk should be screened
            - a brief screening instrument for PTSD should be considered a month after a major disaster
            - refugees and asylum seekers should be screened
- management
    - single debriefing sessions after an event at are best ineffective and at worst harmful
    - for those with mild symptoms, watchful waiting might be appropriate if the event was less than a month ago
    - trauma focused CBT
    - EMDR therapy
    - management of comorbid conditions
    - clonidine is being explored as a potential treatment
    - in severe cases stellate ganglion block may be considered to reduce the action of adrenaline
- prognosis
    - 80-90% recover spontaneously
    - symptoms may still be present years after the event
    - the severity of symptoms two weeks after trauma is a good predictor of severity at six months
    - benefit from treatment does not decline with lapse of time since the event

MHC8 suicide

- key messages from the WHO report on suicide prevention (2014)
    - suicide is a significant health issue
        - over 800,000 people die from suicide every year
        - leading cause of death worldwide in 150=-29 year olds
    - suicides are preventable
        - a comprehensive, collaborative prevention strategy is required
        - communities should be involved and the media should be encouraged to report suicide responsibly
    - restricting access to the means for suicide works

        - including pesticides, firearms and certain medications
    - healthcare services need to incorporate suicide prevention as a core component
    - communities play a critical role in suicide prevention
- risk factors and red flag warning signs
    - demographic and social
        - perception of lack of social support, living alone, no confidants
        - male
        - stressful life events (e.g. recently bereaved, debt/financial worries, loss of attachment/major relationship instability, job loss, moving house)
        - LGBT
        - ethnic minority group
    - personal background
        - substance misuse
        - feeling close to someone who died by suicide or exposure to suicidal behaviour of key others
        - use of suicide-preventing websites or social media
        - access to lethal means
    - clinical factors in history
        - previous self-harm or suicide attempts (regardless of intent, including cutting)
        - mental illness, especially recent relapse or discharge from in-patient care
        - disengagement from mental health services
        - impulsivity or diagnosis of personality disorder
        - long term medical conditions, recent discharge from a general hospital, pain
    - mental state examination and suicidal thoughts
        - high degree of emotional pain and negative thoughts (hopelessness, helplessness, guilt)
        - sense of being trapped/unable to escape and/or a strong sense of shame
        - suicidal ideas becoming worse
        - suicidal ideas with a well-formed plan and/or preparation
        - psychotic phenomena, especially if distressing
- role of the emergency department medical team
    - to complete physical assessment and provide appropriate treatment
    - treat all people in a respectful and non-stigmatising way
    - gather a detailed history of the self-harm including relevant triggers, previous history of self-harm, assessment of mood and level of continuing suicidal intent
    - to gather relevant information about the person's psychiatric history, family and personal circumstances
    - to support the engagement not only of the patient but also, where available, family and friends
    - to consult, and refer where necessary to, mental health colleagues
- safety plans
    - an agreed set of activities, strategies, people and organisations to contact for support if someone becomes suicidal, if their suicidal thoughts get worse or if they might self-harm
        - needs to include explicit reference to removal or mitigation of means of suicide or self-harm

- NICE guidelines recommend that all patients presenting to an acute hospital following an episode of self-harm should receive a psycho-social assessment
- elements of a safety plan:
  - individualised strategies/activities to instil hope
  - calming/distracting activities
  - restriction of access of common means to suicide
  - contacts for social and crisis support
- stayingsafe.net allows a digital safety plan to be made
  - reasons for living and/or ideas for getting through tough times
    - reminders of positive aspects of life – photos of people, pets, special places
    - music that boosts mood and makes people feel good
  - making your situation safer
    - remove things that could be used for self-harm or suicide
    - if stopping self-harm is not an option yet, consider ways to make it safer
    - if medication is in the home, make it safer or store less
    - identify and avoid distress triggers
  - things to lift or calm mood – a calming activity is anything relaxing
    - meditation, yoga, looking at a photo of a great view or someone you care about
    - writing down feelings in a diary or letter
    - calming thoughts such as about a special place or happy memory
  - distractions
    - anything that 'tales your mind away' from distressing feelings
    - distracting activities (e.g. exercise, cooking, art, chores, interacting with someone)
    - distracting via thinking (keeping your mind busy) – e.g. listening to music, crosswords, Sudoku, TV, films)
  - sources of support – anyone you trust
    - day to day support (not necessarily for discussion self-harm/suicide)
    - agree to talk to someone you trust
    - support if distressed or thinking about self-harm or suicide
    - specific suicide and self-harm prevention 24-hour helplines or websites
    - local healthcare support, with emergency NHS contact details for out of hours support

- assessment
  - assess risk factors
    - assessment of mental health
      - past psychiatric history
      - depressive and other psychiatric symptoms
      - medication
      - history of alcohol and illicit drug use
      - observation of verbal and non-verbal indicators of mental state
    - previous self-harm or suicide attempts
    - age, gender, social situation

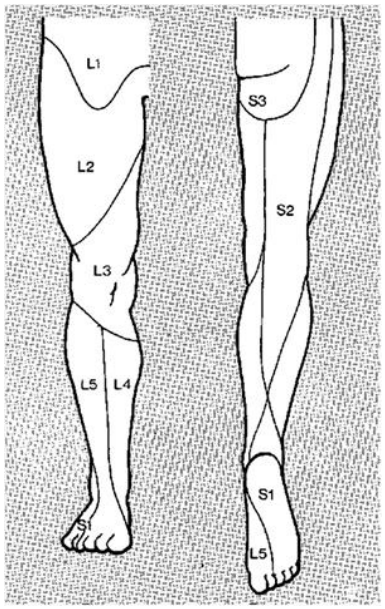
        - relationships which may be supportive/protective and those which pose a threat
        - access to lethal methods
    - assess current intent and plans
        - wish to be dead
        - feelings of hopelessness
        - regret/remorse over current/previous attempts
        - expectation about outcome of self-harming behaviour or suicide attempt/threat
        - specific plans
        - lethality and frequency of plans or attempts
        - other self-harming behaviour
        - assess current suicidal intent/wishes
        - length of time suicidal feelings have been present
        - mental state at time of self-harm or suicide attempt or threat
        - plans for others after death (suicide notes, changes to will, consequences)
    - assess needs
        - social problems
        - untreated mental health disorders
        - physical symptoms and disorders
        - coping strategies
        - skills, strengths and assets
        - psychosocial and occupational functioning
        - personal and financial difficulties
        - needs of dependants
- modified SAD PERSONS scale
    - sex – male = 1
    - age – 15-25 or 59+ = 1
    - depression or hopelessness = 2
    - previous suicide attempts or psychiatric care = 1
    - excessive ethanol or drug use = 1
    - rational thinking loss (psychosis or organic illness) = 2
    - single, widowed or divorced = 1
    - organised or serious attempt = 2
    - no social support = 1
    - stated future intent (determined to repeat or ambivalent = 2
        - 0-5 = may be safe to discharge
        - 6-8 = probably requires psychiatric consultation
        - >8 = probably requires hospital admission

MUSCULOSKELETAL (NON-TRAUMATIC)

MuP1 acute back pain

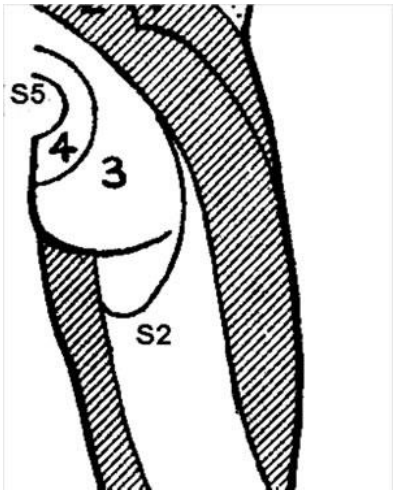
- background
    - 60-80% of people experience back pain at some time in their lives
    - 5-10% have back problems for longer periods
    - around 5% or the population report back problems as chronic sickness

    - EDs see patients with acute back pain, acute exacerbations of a chronic problem and back pain incidental to the presenting problem
- epidemiology
    - back pain tends to start between the late teens and the late 40s/early 50s
    - more common in smokers
    - there seems to be an association with low social class, manual work, low educational achievement
    - lifetime incidence of true sciatica is much less (around 5.3% in men and 3.7% in women)
- history
    - pain history (consider PQRST mnemonic)
        - **P**rovocative and **P**alliative factors
        - **Q**uality of pain
        - **R**adiation
        - **S**everity and **S**ystemic symptoms
        - **T**iming
    - red flags for back pain
        - onset at age <20 or >55
        - non-mechanical pain (unrelated to time or activity)
            - especially if constant and worsening and pain at night
        - thoracic pain
        - previous history of carcinoma, steroids or HIV infection
        - fever, night sweats, weight loss
        - widespread neurological symptoms, especially sphincter disturbance
        - structural spinal deformity
- examination
    - examination of back
    - other examination
        - neurological examination
        - dermatomes
            - including sacral dermatomes (to assess for herniation at L4-S1 nerve root)
        - nerve roots with knee jerk and ankle jerk tendon reflexes
        - ability to straight leg raise
        - observations: heart rate, blood pressure, pulse oximetry and temperature
    - neurological
        - each muscle is normally supplied by more than one nerve root, though one may be dominant
        - myotomes in the lower limb:
            - L2 – hip flexion
            - L3 – knee extension
            - L4 – ankle dorsiflexion, big toe dorsiflexion
            - L5 – foot inversion
            - S1 – ankle plantar flexion
        - dermatomes in the lower limb:



https://www.rcemlearning.co.uk/reference/lower-back-pain/#1567605105406-4cb2d1ad-66f9

- tendon reflexes
    - knee jerk (L3, L4)
    - ankle jerk (S1, S2)
- straight leg raise
    - many patients with back pain have pain radiating to the buttock or leg
    - in true sciatica (commonest lesions at L5/S1), pain radiates below the ankle
    - some patients with nerve root problems only have pain in the leg and not the back
    - patients will have pain if the sciatic nerve is stretched
        - straight leg raise
        - dorsiflexion of foot while leg is raised
- features of nerve root lesions
    - paraesthesia localised to the dermatome of the involved nerve root
    - altered sensation in the same dermatome
    - muscle weakness in the corresponding myotome
    - muscle weakness on one side with sensory changes on the other side or dissociated sensory changes (e.g. loss of light touch on one side and pain sensation on the other side) are serious signs suggesting involvement of the spinal cord rather than a nerve root
- rectal examination and testing of the sacral dermatomes is essential



https://www.rcemlearning.co.uk/reference/lower-back-pain/#1567605105406-4cb2d1ad-66f9

- differential diagnosis
    - structural
        - mechanical or non-specific
        - facet joint arthritis or dysfunction
        - prolapsed intervertebral disc
        - annular tear of intervertebral disc
        - spondylolysis or spondylolisthesis
        - spinal stenosis
    - neoplasm
        - primary or secondary including multiple myeloma
    - referred pain to spine from:
        - major viscera
        - retroperitoneal structures
        - aorta
        - hip
    - infection
        - discitis
        - osteomyelitis
        - paraspinal abscess
    - inflammatory
        - spondyloarthropathies
        - sacroiliitis or sacroiliac dysfunction
    - metabolic
        - osteoporotic vertebral collapse
        - Paget's disease
        - osteomalacia
        - hyperparathyroidism
    - causes of back pain
        - cauda equina syndrome
        - non-traumatic compression fractures
            - 1 in 3 lifetime risk for women over 50 of vertebral compression fracture (including asymptomatic fractures)
            - may be difficult or impossible to date a fracture on x-ray

        - if there is no significant history and the patient presents with acute back pain, it is safe to assume that the fracture is new/recent
    - postmenopausal women with a fracture are at much greater risk of subsequent fractures
    - other things to consider in patients with non-traumatic fractures are metastases and multiple myeloma
- spondylolisthesis
    - forward slippage of one vertebra on another
    - most common sites are L5 on S1 and L4 on L5
    - usual cause for L5 slippage is a defect of the pars inter-articularis of L5
        - may be congenital or traumatic, tends to occur in younger people
    - slippage of L4 on L5 is usually degenerative
    - may be an incidental finding, so its discovery does not necessarily mean it is the cause of the patient's back pain
    - CT may be helpful in determining exact cause and age of spondylolisthesis
- infection
    - uncommon but must be considered
    - spinal TB should be particularly considered in ethnic minority groups
    - most common presenting symptom is back pain and there may be spinal tenderness or kyphosis
    - may present with a paraspinal abscess as loin swelling or as a psoas abscess with spasm and hip flexion
    - neurological symptoms can also occur
    - systemic symptoms such as weight loss and night sweats can occur but is not common in bony TB
    - no evidence as to whether CT or MRI is preferable
- malignancy
    - signs and symptoms of metastasis include:
        - pain in the thoracic or cervical spine
        - progressive lumbar spinal pain
        - severe unremitting lower spinal pain
        - spinal pain aggravated by straining (e.g. passing stool, coughing, sneezing)
        - localised spinal tenderness
        - nocturnal spinal pain preventing sleep
    - investigation of choice is MRI

- investigations
    - no investigation required for most back pain
        - imaging of patients with non-specific back pain and no red flags is unhelpful
        - patients with pain may have normal imaging, whereas patients with abnormal imaging may have no pain
    - red flags for cauda equina require urgent MRI
    - blood tests are only useful if infection or metabolic problems are suspected

- in primary care, x-rays for patients with pain ongoing for longer than 6 weeks improved patient satisfaction but not outcome

- management
  - symptomatic treatment of acute musculoskeletal lower back pain
    - analgesia
      - NSAIDs slightly more effective than placebo for short term symptomatic relief
    - muscle relaxants
      - can be effective in the management of non-specific low back pain but adverse effects require caution in their use
    - activity
      - patients should be encouraged to remain active
      - many people get some relief by lying down but bed rest is less effective in reducing pain and improving ability to perform ADLs
    - physiotherapy
      - value of physiotherapy does not have a good evidence base, but there are varied forms some of which may be helpful
    - other treatments that have been investigated include:
      - traction
        - no more effective than placebo as single treatment
      - massage
        - not enough evidence for acute pain
        - is of value for chronic pain
      - antidepressants
        - common treatment
        - can provide pain relief, help with sleep and reduce depression
        - a Cochrane review found no benefit over placebo, and they have side effects
        - may have a role in neurological pain in sciatica
      - local heat
        - moderate evidence that heat wrap therapy reduces pain and disability for patients with back pain that lasts less than 3 months
        - relief occurs only for a short time and the effect is small
        - addition of exercise to heat wrap therapy appears to provide added benefit
      - local cold
        - not enough evidence available
      - individual patient education
        - patients who had an in-person education session lasting at least 2 hours in addition to usual care had better outcomes
        - shorter education sessions or written information without in-person education did not provide additional benefit
      - spinal manipulative therapy
        - more effective than sham therapy in reducing pain and improving ability to perform ADLs, but no more or less

effective than medication, physical therapy, exercises, back school or the care given by a GP
                - exercise therapy
                    - not shown to improve pain or function over no treatment or conservative therapies
                    - seems to be slightly effective for chronic back pain
                    - a graded activity program may reduce absenteeism in subacute low back pain
                - lumbar supports
                    - unclear whether these are more effective than no or other treatments
                - acupuncture
                    - no evidence to support its use
                - strong opiates
                    - no published trials to look at this
    - treatment of sciatica
        - epidural injection therapy is often used, but its value has not been proven
        - surgical discectomy for carefully selected patients with sciatica due to a prolapsed lumbar disc appears to provide faster relief than non-surgical management
            - longer term outcomes are unclear
            - microdiscectomy gives broadly similar results
    - treatment of vertebral compression fractures
        - conventional treatment is not evidence based and revolves around analgesia to relieve symptoms
        - spinal support may help but patients also need to mobilise to regain back movement
        - majority of patients become symptom free through these measures
        - patients who continue to have pain should have repeat lateral standing lumbar spine x-rays to look for progressive deformity
        - surgery may be considered when there is continued vertebral collapse and severe pain
            - they may also be helped by less invasive procedures, including balloon kyphoplasty
        - osteoporosis will need to be investigated and managed
    - treatment of metastatic disease
        - patients with bone metastases or those at risk of them should be given information explaining what to do and who to contact if they develop symptoms of spinal metastases or spinal cord compression or if symptoms worsens whilst awaiting investigation
        - spinal cord compression is an oncological emergency and treatment should be started within 24 hours
            - most patients will be given steroids and surgery or radiotherapy
            - patients with a risk of spinal instability should be nursed flat in neutral alignment

        - NICE recommends that every centre has a co-ordinator for patients with spinal metastases and metastatic spinal cord compression and patients should be referred to them
- follow up and prognosis
    - follow up, if required, is not the role of the emergency physician but may be required by GPs, orthopaedic surgeons, spinal surgeons, geriatricians or rehabilitation teams
    - prognosis is good for non-specific musculoskeletal back pain
        - pain and disability normally decrease rapidly within a month and continue to improve until about 3 months
        - there is a 73% chance of recurrence within a year
    - predictors of poor outcome (in a primary care study) include:
        - premorbid factors
            - increasing age
            - female gender
            - previous history of back pain
            - high levels of psychological distress
            - poor self-rated health
            - smoking
            - dissatisfaction with employment
        - factors relating to back pain
            - duration of symptoms
            - pain radiating to the leg
            - widespread pain
            - restriction in spinal mobility
        - patients with sciatica return to work more slowly than those with non-specific pain

MuP2 limb pain and swelling

- limb pain by site
    - upper
        - shoulder and upper arm
            - traumatic/acute
                - shoulder dislocation
                    - anterior/posterior/inferior
                - clavicle fracture
                - humerus fracture
                - scapula fracture
                - acromioclavicular joint injury
                - glenohumeral instability
                - rotator cuff tear
                    - rotator cuff tests
                        - supraspinatus test
                            - abduct arm to 90 degrees, forward flex it 30 degrees with thumb down (‘empty beer can’ position)

        - test for pain/weakness against resistance to continued abduction
    - infraspinatous and teres minor test
        - stabilise the elbow against the patient's waist and bend the elbow to 90 degrees
        - test for pain/weakness against resistance to external rotation
    - subscapularis
        - place hand behind lower back
        - test for pain/weakness as patient attempts to push examiner's hand away by moving dorsum of hand away from back
    - drop arm test
        - patient is unable to hold or smoothly lower an extended arm at 90 degrees of shoulder abduction without dropping it
  - biceps tendon rupture
  - triceps tendon rupture
  - septic joint
- non-traumatic/chronic
  - rotator cuff tear
  - impingement syndrome
    - impingement tests
      - manoeuvre of Neer
        - prevent scapular rotation with one hand while raising patient's straightened arm in full forward flexion to overhead
        - positive sign is pain in the arc between 70 and 120 degrees
      - Hawkins impingement test
        - position the shoulder at 90 degrees of abduction and elbow at 90 degrees of flexion
        - then rotate shoulder internally, bringing the arm across the front of the patient
        - positive sign is pain during this manoeuvre
  - calcific tendinitis
  - adhesive capsulitis
  - biceps tendinitis
  - subacromial bursitis
  - cervical radiculopathy

- referred pain and non-orthopaedic causes
    - referred pain
        - neck
        - diaphragm
    - brachial plexus injury
    - axillary artery thrombosis
    - thoracic outlet syndrome
    - subclavian steal syndrome
    - Pancoast tumour
    - myocardial infarction
    - pneumonia
    - pulmonary embolism
- elbow
    - radiograph-positive
        - distal humerus fracture
        - radial head fracture
        - capitellum fracture
        - olecranon fracture
        - elbow dislocation
    - radiograph-negative
        - lateral epicondylitis
        - medial epicondylitis
        - olecranon bursitis
        - septic bursitis
        - biceps tendon rupture/dislocation
    - paediatric
        - pulled elbow
        - supracondylar fracture
        - lateral epicondyle fracture
        - medial epicondyle fracture
        - olecranon fracture
        - radial head fracture
        - Salter-Harris fractures
- forearm
    - distal radius fracture
        - Colles'/Smith/Barton/radial styloid/distal radioulnar joint disruption
    - radio-ulnar fracture
    - isolated radius fracture
    - isolated ulna fracture
    - Monteggia fracture dislocation
    - Galeazzi fracture dislocation
    - carpal fractures

- lower
    - thigh/upper leg
        - hip pain
            - acute trauma

        - femur fracture
            - proximal
                - intracapsular
                    - femoral head fracture
                    - femoral neck fracture
                - extracapsular
                    - intertrochanteric femoral fracture
                    - trochanteric femoral fracture
            - midshaft femur fracture
        - hip dislocation
        - pelvic fractures
            - acetabular
            - open book
            - straddle
            - avulsion
    - chronic/atraumatic
        - hip bursitis
        - psoas abscess
        - piriformis syndrome
        - meralgia paresthetica
        - septic arthritis
        - obturator nerve entrapment
        - avascular necrosis of hip
- knee
    - acute/traumatic
        - knee dislocation
        - knee fracture
            - patella
            - tibial plateau
        - meniscus and ligament knee injuries
        - patella dislocation
        - patella tendonitis
        - patella tendon rupture
        - quadriceps tendon rupture
    - nontraumatic/subacute
        - arthritis
        - gout/pseudogout
        - Osgood-Schlatter disease
        - patellofemoral syndrome (runner's knee)
        - patellar tendonitis (jumper's knee)
        - pes anserine bursitis
        - popliteal cyst (Baker's cyst)
        - prepatellar bursitis
        - septic bursitis
        - septic joint

        - DVT
    - lower leg
        - fractures
            - tibial plateau fracture
            - tibial shaft fracture
            - Pilon fracture
                - tibial plafond driven into talar dome, high force
            - Maisonneuve fracture
                - lower extremity equivalent of Galeazzi fracture, caused by external rotation applied to foot
            - ankle fracture
        - calf pain
            - Achilles' tendon rupture
            - calcaneal bursitis
            - cellulitis
            - compartment syndrome
            - DVT
            - gastrocnemius strain
            - ruptured popliteal cyst
            - superficial thrombophlebitis

MuP3 neck pain

- background
    - common and potentially life-threatening causes
        - acute wry neck/torticollis
        - spondylosis
        - disc impingement
        - spinal stenosis
        - meningitis
    - uncommon but life-threatening causes
        - pharyngitis
        - infective discitis
        - spinal osteomyelitis
        - metastases
    - risk stratification
        - most patients with non-traumatic neck pain do not require imaging
            - imaging is required if there are neurological findings
        - high risk patients include:
            - insidious onset of symptoms
            - significant comorbidities
                - diabetes
                - immunosuppression
- differentiating between musculoskeletal pain and radiculopathy
    - musculoskeletal
        - pain is a deep, dull, episodic ache

        - history of excessive or unaccustomed activity
        - localised and asymmetric pain
        - referred pain: head, limb girdle
        - symptoms aggravated by neck movement, relieved by rest
    - radiculopathy
        - pain is sharp or burning
        - radiates to trapezial and periscapular areas or down the arm
        - numbness/weakness in myotomal distribution
        - headache may occur if upper cervical roots are involved
        - symptoms aggravated by neck hyperextension (especially when head is towards affected extremity)
        - gradual onset of shocklike sensations spreading down spine to extremities
        - most common at C5 (shoulder abduction, external rotation)
- related pathophysiology
    - radiculopathy
        - referred pain into the upper extremities often accompanies neck pain
        - referred pain can be the initial symptom of a compressed nerve root by a ruptured disc or stenosis at the foramina from osteophytes
        - radicular pain is described as sharp or shock-like
            - may be associated with certain activities or positions
            - distribution of pain usually follows dermatomes
        - sensory changes are often seen, with numbness and tingling common and decreased sensation in a dermatomal pattern
        - motor weakness may be present
            - longstanding motor deficits may be associated with wasting
            - hyporeflexia may be seen
        - cervical radiculopathy
            - can present acutely (e.g. traumatic ruptured disc) or more chronic and intermittent (e.g. foraminal narrowing from osteophytes)
            - inferior nerve root is typically affected (e.g. C5-C6 disc abnormalities affect the C6 nerve root)
            - C5-6 and C6-7 are most commonly affected
            - C5 radiculopathy generally presents with pain in the shoulder and the upper part of the lateral arm
                - paraesthesia is often seen more in the distal part of the affected dermatome
                - deltoid weakness is commonly seen
            - C6 radiculopathy often has biceps or brachioradialis weakness and hyporeflexia
                - paraesthesias and sensory loss can extend into the hand
            - C7 root compression causes triceps weakness and decreased triceps reflex
                - pain into the distal forearm or hand is common
            - cervical radiculopathy often has a history of improvement with time (slow, spontaneous recovery)
                - disc herniation may be less likely to improve spontaneously
            - non-surgical therapies include:

                - oral or epidural steroids
                - cervical traction
                - physical therapy
                - bracing
            - surgical management is considered for some
        - myelopathy
            - the clinical presentation of pathology affecting spinal cord function
            - large differential for causes of myopathy, including:
                - trauma
                - metabolic
                - degenerative
                - inflammatory
                - toxic
                - infectious
                - neoplastic
            - longstanding myelopathy is rarely reversible
                - early identification is important to prevent loss of neurological function
            - the most general signs are those of upper motor neurone dysfunction
                - subtle symptoms include:
                    - difficulty with fine motor control of hands and fingers
                    - gait problems and instability
                    - numbness
            - there is hyperreflexia, increased tone and weakness
            - patients presenting with acute onset myelopathy need urgent MRI
            - progression can be slow and gradual or stepwise
                - patients may not seek diagnosis until late due to insidious onset
- clinical assessment of neck pain
    - history
        - red flags
            - presentation in patients under 20 or over 55
            - constant, progressive pain
            - past history of carcinoma
            - systemic steroids
            - drug abuse
            - HIV
            - systemically unwell
            - weight loss
            - persisting severe restriction of cervical flexion
            - inflammatory disorders such as ankylosing spondylitis and rheumatoid disease
- specific conditions
    - acute wry neck/torticollis
        - commonly affects adolescents and young adults

        - patient presents with the neck held at an angle, in constant pain
        - pain tends to be localised to mid cervical region and is unilateral away from the direction of the deformity
        - patient often describes a sudden unguarded movement of the neck causing pain and restricted neck movement
        - normally no history of trauma
        - management:
            - NSAIDs
            - heat
            - time
        - pathology is unclear – possibly nipped/trapped synovium or mechanical sticking of roughened surfaces
    - spondylosis
        - very common in over 50s
        - usually causes no significant symptoms
        - minor injury such as a missed step can cause an exacerbation resulting in pain
            - pain appears disproportionate to the trivial injury
        - radiographic features are osteophytic lipping and narrowed disc space (seen at C6/C7 below)

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    - disc impingement
        - in cervical disc prolapse part of the nucleus pulposus can protrude through the annulus fibrosis at the weakest part, most commonly posterolateral
        - a slight protrusion bulges against the posterior longitudinal ligament and causes localised neck pain
        - if larger, it may impinge upon the spinal nerve root at that level or the spinal cord itself
        - may occur in younger people due to injury or older people as a result of degeneration and cervical spondylosis

        - when pain is severe and unremitting, associated with radicular features, disc impingement should be suspected
        - neck pain associated with degenerative disc disease and osteophytes will improve in most people without invasive treatment, although some go on to have chronic symptoms
    - spinal stenosis
        - results in myelopathy
        - usually progresses slowly and can initial be subtle
        - most common presenting complaints include neck pain, gait problems, hand numbness and clumsiness
        - loss of bladder and bowel control is uncommon in the early stages
        - occasionally patients present with acute and profound spinal cord dysfunction after a hyperextension injury
            - a stepwise decline is more common
        - typical patients are male and over 50
        - examination findings include:
            - increased reflexes in the upper and lower extremities
            - Babinski reflex
            - Lhermitte's sign occurs in a minority
                - electric shock-like pain radiating down the spine on neck flexion
            - lower motor neurone features occurring secondary to nerve root compression such as wasting, fasciculations, hypoactive reflexes
        - differentials include:
            - multiple sclerosis
            - syringomyelia
            - spinal cord tumour
            - subacute combined degeneration
            - normal pressure hydrocephalus
        - if there is myelopathy and any significant canal stenosis, surgical decompression is recommended
            - this does not normally completely correct any deficits so decompression is often offered early
            - operative indications are less clear when there is significant stenosis without signs or symptoms or myelopathy
    - meningitis
        - often creates a typical board-like rigidity of the neck
        - fever will usually be present, and possibly photophobia and headache
        - time course is acute
    - pharyngitis
        - may cause neck pain
        - has a peak in late summer
        - usually viral cause
        - Lemierre's syndrome
            - rare but life threatening pharyngitis in young adults
            - usually caused by an anaerobic bacterium, Fusobacterium necrophorum

        - characterised by oropharyngeal infection with evidence of septic thrombophlebitis
        - managed with metronidazole and clindamycin as first line
- peritonsillar abscess
    - usually associated with ipsilateral ear pain and obvious swallowing difficulty
    - usually progresses from tonsillitis to cellulitis and ultimately abscess formation
        - probably involvement of Weber's glands – mucous salivary glands superior to the tonsil in the soft palate which clear the tonsil of debris
        - tissue necrosis and pus formation occurs when they become inflamed, most commonly just above the superior pole of the tonsil
- Ludwig's angina
    - infection of the tissues of the floor of the mouth, usually alongside a dental infection
    - usually Streptococcus
    - usual route of infection is from infected lower third molars or from infection of the gums surrounding the lower third molars
    - usually occurs in immunosuppressed patients but can happen in otherwise healthy people
- infective discitis and spinal osteomyelitis
    - infective discitis usually occurs in the immunocompromised, often IV drug users
        - usually causes fever
        - may progress to lytic collapse of the vertebral body with catastrophic quadriplegia
        - onset is insidious and diagnosis is difficult
        - there may be a distant focus of infection with haematological spread to the spinal column
            - skin and genitourinary tract are common antecedent sites
            - there may be multiple foci
        - most common organism is Staphylococcus aureus but Pseudomonas species are common in IV drug users
- nonpyogenic osteomyelitis can be caused by tuberculosis, fungus, yeast or parasitic organisms
- 30-70% of patients with vertebral osteomyelitis have no obvious prior infection
    - risk factors include immunocompromising conditions such as:
        - advanced age
        - IV drug use
        - congenital immunosuppression
        - long-term systemic steroids
        - diabetes
        - organ transplantation
        - malnutrition
        - cancer
    - x-rays may show no abnormality early on but ESR and CRP are likely to be raised; bone scintigraphy is likely to be abnormal

- metastases
    - deposits have a similar radiological appearance to the lytic lesions of infection
    - in many cases there is no known history of carcinoma

- investigations
    - plain x-ray
        - requires 3 films of the c-spine: lateral, AP and open mouth (peg) views
        - ABCD system of review:
            - adequacy/alignment
                - entire cervical spine should be visible from the occipital condyles to the top of the T1 vertebral body
                    - if inadequate, it may be possible to repeat the view with someone pulling caudally on the arms, or local policy may be to proceed to CT
                - adequate exposure of the spinous processes and the soft tissues anterior to the vertebral bodies
                - four lines:
                    - anterior aspect of the vertebral body, marking the line of the anterior longitudinal ligament
                    - posterior aspect of the vertebral body, marking the line of the posterior longitudinal ligament and the anterior limit of the spinal canal
                    - spino-laminar line at the junction of the laminae and the spinous processes – posterior limit of the spinal canal
                    - spinous process line

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            - bones
                - assessment of each bone for injury or structural abnormalities
                - otontoid process should be scrutinised from fractures, which occur most commonly at the base
                    - the Mach effect may resemble a fracture when there is superimposition of normal structures (usually the base of the skull)
                - next assess the body of C2 and the integrity of C1

        - there should be no more than 12mm between a line drawn from the tip of the basion (anterior edge of the foramen magnum) to the tip of the dens
        - the body of C2 should have a visible ring on the lateral view (the Harris ring)
            - may be incomplete between the 5 and 7 o'clock position but disruption elsewhere should raise suspicion of a fracture
    - cartilage
        - cartilage is not visible on plain imaging but the cartilaginous spaces should be assessed for uniformity
        - special note of:
            - distance between the vertebral bodies from C2/3 onwards
                - should be similar at all levels
            - the C1/2 articulation
                - distance between the anterior margin of the odontoid process and the posterior portion or the arch of C1 should be no greater than 3mm in the adult spine
            - the facet joints
                - assess for uniformity at each level
    - dense soft tissues
        - paravertebral soft tissues – evidence of oedema or haematoma
        - soft tissue between the larynx and spine for swelling or distortion
            - possible infection or underlying fracture
        - there may be an apparent increase in the soft tissue anterior to C1 and C2 in flexion, particularly in young children
- variations in paediatric patients:
    - relatively large head leading to higher fulcrum of flexion (C2/C3)
    - horizontally aligned facet joints
    - underdeveloped uncinate process leading to flatter articular surface
    - anterior wedging of vertebral bodies
    - cartilaginous synchondrosis at the junction of the odontoid peg and C2 vertebral body
    - less rigid ligamentous support and weak supportive muscles

- CT
    - useful for suspected bony lesions
    - in spontaneous neck pain, if there is suspicion of spondylotic change, metastases or osteomyelitis it is the first choice of investigation
- MRI
    - T1 weighting has an increased signal from fatty tissue and a lower signal from fluid, giving a more structural view
        - highlights bony destruction or infiltration with loss of fatty tissue
    - T2 weighting produces images containing deep muscle tones and bright fluid

                - particularly useful to investigate cord or disc lesions
        - myelography
            - injection of contrast medium into the cervical space with fluoroscopic guidance followed by x-rays
            - largely replaced by CT and MRI but may help to find causes of pain not seen on CT or MRI
            - CT or MR can be performed after myelographic material has been placed and CT myelogram is most useful for patients who cannot have MRI (e.g. pacemaker, cochlear implant)
- management
    - adequate pain relief with combination analgesia therapies
    - confident diagnosis
    - seek specialist opinion for unusual cases
    - physiotherapy
        - aims to reduce pain, improve posture and improve range of movement
        - treatments often include manual mobilisation of joints segmentally

MuP4 joint swelling

- differential of acute monoarthritis
    - septic arthropathy
        - bacterial
            - e.g. streptococcal, staphylococcal
        - viral
            - e.g. mumps. EBV, hepatitis B, enteroviruses
                - may cause synovial infection or reactive arthritis
        - fungal
        - mycobacteria
        - Lyme disease
        - brucellosis
        - leptospirosis
    - crystal arthropathy
        - gout (uric acid)
        - pseudogout (calcium pyrophosphate)
        - apatite arthropathy
        - calcium oxalate arthritis
    - bony or cartilaginous disease
        - avascular necrosis
        - osteochondritis dissecans
        - ligamentous injury/instability or soft tissue injury
        - osteoarthritis
        - osteomyelitis
        - overuse injury
        - loose body in joint
        - bone tumour or metastasis
    - inflammatory arthritis
        - rheumatoid arthritis with monoarthritic presentation
        - juvenile idiopathic arthritis/adult onset Still's disease

        - vasculitis
        - associated with inflammatory bowel disease
        - relapsing polychondritis
        - psoriatic arthropathy
        - pigmented villonodular synovitis
    - manifestation of systemic illness
        - sarcoidosis/systemic lupus erythematosus
        - rheumatic fever
        - Behçet's disease
        - reactive arthritis
        - hypertrophic pulmonary osteoarthropathy
        - ankylosing spondylitis
        - familial Mediterranean fever
        - amyloid arthropathy
    - trauma or haemorrhage
        - peri-articular or intra-articular fracture
        - traumatic effusion
        - haemarthrosis
        - associated with haemoglobinopathy
        - neuropathic joint (painless)
- investigations may include:
    - aspiration
        - unless overlying cellulitis, raised INR, prosthetic joint or other contraindication
        - synovial fluid changes:
            - normal
                - clear, viscous fluid
                - WBC 0-200
                - no crystals
                - sterile culture
            - septic arthritis
                - turbid, low viscosity
                - WBC 50,000 – 200,000
                - no crystals
                - positive culture in some cases
            - gout
                - clear, low viscosity
                - WBC 500-200,000
                - needle shaped, negatively birefringent crystals
                - sterile culture
            - pseudogout
                - clear, low viscosity
                - WBC 500-100,000
                - block shaped, positively birefringent crystals
                - sterile culture
            - inflammatory
                - turbid, yellowish-green (chicken soup)

        - WBC 2,000-100,000
        - no crystals
        - sterile culture
    - osteoarthritis/injury
        - large volume, normal viscosity, may be blood stained if trauma/haemoarthrosis
        - WBC 0-2,000
        - usually no crystals (5% have pyrophosphate crystals)
        - sterile culture
- urinalysis
    - may be indicated for microscopic haematuria/protein in inflammatory conditions
- bloods
    - cultures if sepsis suspected
    - FBC, ESR, CRP, U&E may be helpful
    - consideration of rheumatoid factor and autoantibodies if inflammatory arthritis suspected
    - antistreptolysin O (ASO) titre and throat swab if rheumatic fever a possibility
- x-rays
    - often normal/unhelpful
    - may reveal evidence of gout or other underlying pathology

MuP5 acute hot swollen joint

- symptoms
    - sudden or gradual onset of joint swelling
        - septic arthritis and crystal arthropathies tend to present acutely (typically within two weeks)
        - rheumatoid arthritis and osteoarthritis have a more insidious onset
        - sudden onset swelling or excessive swelling of one joint compared with others in patients with known inflammatory arthritis should raise the suspicion of septic arthritis
    - number of joints involved
        - gout and septic arthritis usually present as a monoarthritis (although more than one joint can be involved
        - reactive arthritis and rheumatoid arthritis tend to affect several joints
    - joints affected
        - the most usual joints for septic arthritis are the knee in adults and the hip in children, although any joint can be affected
        - in gout up to 70% of first attacks are in the big toe
            - it can occur elsewhere in the foot as well as the ankle, knee, wrist, elbow and small joints of the hands
            - atypical presentations are more common in patients over 60 years
    - constitutional symptoms
        - patients with septic arthritis may have general malaise and a history of fevers and rigors
            - however systemic symptoms occur in less than half of adults with confirmed septic arthritis

                - systemic symptoms may point to the onset of septic arthritis in someone with inflammatory arthritis
            - a history of gastrointestinal or genitourinary infection raises the possibility of reactive arthritis
            - Lyme disease is often accompanied by general symptoms of fever, malaise, arthralgia, myalgia and headache and is worth considering in patients with potential exposure to deer ticks in an endemic area
        - history of trauma
            - haemarthrosis usually develops rapidly after an episode of trauma and results in a swollen painful joint
            - check for any recent animal or human bite over the affected joint as a puncture wound can be the source of septic arthritis
- signs
    - general appearance of the patient and any pyrexia
    - inspect the joints for swelling, redness and any deformity
    - redness is relatively uncommon and should raise suspicion of septic arthritis or gout
    - warmth suggests an inflammatory process
        - beware of a recently removed bandage or application of a warm compress
    - swelling
        - due to effusion of joint or oedema in the surrounding tissues
        - swelling over a bursa or tender suggests inflammation of that structure
        - bony swelling would be in keeping with osteoarthritis
    - tenderness
        - elicited by gentle palpation over the joint
        - point tenderness may be due to inflammation in periarticular soft tissue and suggests bursitis or soft tissue injury
    - range of movement
        - passive and active range of movement is normally significantly reduced in septic arthritis
    - signs of joint disease
        - e.g. rheumatoid nodules or gouty tophi
- differential diagnosis
    - infection
        - septic arthritis
        - Neisseria gonorrhoeae
        - Lyme disease
    - rheumatoid arthritis
    - crystal arthropathies – gout and pseudogout
    - reactive arthritis
    - trauma
    - palindromic rheumatism
        - multiple and unpredictable episodes of arthritis which can be severe, affecting different joints at different times
        - joints tend to return to normal between attacks
    - psoriatic arthropathy
- investigations
    - joint aspiration
        - required if septic arthritis is suspected

        - warfarin is not a contraindication
    - synovial fluid is often turbid and purulent but the absence of pus does not exclude infection
    - fluid should be sent for Gram staining and culture before starting antibiotics
        - absence of organisms does not exclude infection
    - examination of crystals to exclude gout/pseudogout
    - synovial fluid white cell count and percentage of polymorphonuclear cells may indicate need for treatment for septic arthritis before Gram staining and cultures are available
- laboratory tests
    - FBC
        - raised WCC is in keeping with infection but can also be raised in crystal arthropathies
        - not always a reliable sign of septic arthritis, particularly in children
    - inflammatory markers
        - ESR and CRP are generally higher in septic arthritis than in gout but cannot be relied upon for diagnostic purposes
        - useful markers of response to treatment
    - electrolytes and liver function
        - end organ damage may affect antibiotic choice
    - urate
        - not a specific test – hyperuricaemia often occurs in patients without gout
        - uric acid levels can fall to within normal ranges during an attack of gout and should not be used as an acute diagnostic test
        - urate levels can be useful for monitoring medication between attacks
    - rheumatoid factor
        - should only be requested if there are strong clinical suspicions of systemic rheumatic disease
        - may be appropriate in the context of polyarthritis
    - antinuclear antibodies (ANAs)
        - should not be routinely requested but can be considered for suspected connective tissue disease
        - tends to be positive in most lupus patients and can be raised in rheumatoid arthritis
    - blood cultures
        - should always be taken in cases of suspected septic arthritis
- imaging
    - x-rays
        - plain film if history of trauma
        - debatable diagnostic value in septic arthritis but may show evidence of chondrocalcinosis
        - often normal in the acute setting in gout apart from soft tissue swelling
            - later erosive changes and tophi may be seen
    - MRI
        - can identify osteomyelitis

    - US
        - may be required to assist in aspiration of a suspected septic hip
    - CT
        - may be used to assist in difficult aspirations or where cartilage or bony abnormalities need to be visualised
- management
    - antibiotics after aspiration and urgent orthopaedic review for suspected septic arthritis
    - other management as per diagnosis

MuC1 cauda equina syndrome

- background
    - the cauda equina is formed by nerve roots caudal to the level of spinal cord termination
    - cauda equina syndrome is caused by compression of the nerves, causing one or more of bladder and/or bowel dysfunction, reduced sensation in the perineum and sexual dysfunction with possible neurological deficit in the lower limbs
- causes
    - herniation of a lumbar disc
        - most commonly central disc herniation at L4/L5 and L5/S1
    - tumours
        - metastases
        - lymphoma
        - spinal tumours
    - trauma
    - infection, including epidural abscess
    - congenital
        - e.g. congenital spinal stenosis, kyphoscoliosis, spina bifida
    - spondylolisthesis
    - late stage ankylosing spondylitis
    - postoperative haematoma
    - following spinal manipulation
    - inferior vena cava thrombosis
    - sarcoidosis
- symptoms and signs are:
    - acute low back pain
        - may be superimposed on a history of chronic back problems
    - radiation of pain to the legs
        - usually, but not always, bilateral
    - lower limb weakness
        - frequently bilateral
    - alteration of sacral and perineal sensation
        - usually but not always bilateral
    - alteration of bladder and/or bowel habit leading to urinary retention and constipation
- cautions
    - not all patients have signs

        - the most sensitive are low back pain, perineal and sacral sensory loss and bladder disturbance
        - it is possible to have bladder dysfunction in the presence of normal anal tone
            - may be missed if not considered and examined for
        - low threshold for requesting MRI
            - cauda equina is an emergency that must be operated on within 24 hours of bladder symptoms appearing
                - when the patient attends the ED they may already have had bladder symptoms for some time
- investigations
    - MRI
- differential
    - conus medullaris syndrome
        - located above the cauda equina at T12-L2
        - nerve root pain is less prominent and the main features are urinary retention and constipation
    - mechanical back pain and prolapsed lumbar disc
    - fracture of lumbar vertebrae due to trauma
    - spinal tumour
    - spinal cord compression
    - peripheral neuropathy
- management
    - urgent neurosurgical referral
    - spinal immobilisation if due to trauma
    - surgery
    - radiotherapy if malignant disease as cause and surgery not an option
    - postoperative care includes addressing lifestyle issues (e.g. obesity), physiotherapy, occupational therapy
- prognosis
    - depends on aetiology and time taken before effective management
    - inadequate or delayed diagnosis and treatment often lead to long-term bladder, bowel and sexual dysfunction
    - patients with bilateral sciatica or complete perineal anaesthesia have a less favourable prognosis than patients with unilateral pain

MuC2 crystal related arthropathies

- gout
    - definition
        - can be defined as arthritis due to deposition of monosodium urate crystals within joints causing acute inflammation and eventual tissue damage
    - background
        - duration and magnitude of hyperuricaemia is directly correlated with the likelihood of developing gouty arthritis and developing uric acid kidney stones
            - gout can however occur in people with normal plasma urate levels and many people with hyperuricaemia never develop gout

        - hyperuricaemia is usually due to impaired renal excretion of urate
        - around 90% of people with hyperuricaemia are under-excretors of urate and around 10% are over-producers of urate
            - some are both
            - the cause is often multifactorial
    - epidemiology
        - more common in men and in older people
            - only 3-6% have the onset before 25 years of age
            - male: female ration around 3.6:1
        - may be more common in some non-white ethnic groups
    - risk factors
        - male gender
        - meat
        - seafood
        - alcohol (10 or more grams/day)
        - diuretics
        - obesity
        - hypertension
        - coronary heart disease
        - diabetes mellitus
        - chronic kidney disease
        - high triglycerides
        - heart failure
        - psoriasis
        - chemotherapy
    - presentation
        - EULAR guidelines for diagnosis suggest that a highly typical presentation is development of acute pain in a joint which becomes swollen, tender and erythematous and which reaches its crescendo over 6-12 hours
        - 50% of attacks and 70% of first attacks affect the first MTP
        - other sites often affected are:
            - knee
            - midtarsal joints
            - wrists
            - ankles
            - small hand joints
            - elbows
        - inflammation reaches its peak within 24 hours, often with fever and malaise
        - some patients may present only with connective tissue tophi
    - signs
        - florid synovitis and swelling and extreme tenderness with overlying erythema
        - untreated, the attack resolves in 5-15 days, usually with itching and desquamation of overlying skin
        - atypical attacks can occur with tenosynovitis, bursitis and cellulitis, with mild discomfort without swelling lasting a day or two
        - chronic tophaceous gout

- large crystal deposits produce irregular firm nodules mainly around extensor surfaces of the fingers, hands, forearms, elbows, Achilles tendons and ears
- typically asymmetrical with a chalky appearance beneath the skin
- damage is usually found in the first MTP joints, midfoot, small finger joint and wrist, with restricted movement, crepitus and deformity

- investigations
  - typical presentations such as inflammation of the first MTP joint with hyperuricaemia can be clinically diagnosed – but not definitive unless the presence of uric acid crystals can be confirmed
  - demonstration of MSU crystals in synovial fluid or tophi confirms diagnosis
  - all synovial fluid should be examined for crystals as gout can present atypically, and Gram staining/culture should be done as gout and septic arthritis can co-exist
  - the cutoff point at which uric acid is considered raised is 360 μmol/L
  - renal uric acid secretion (24-hour urine collection) may be helpful in diagnosis, particularly in young onset and patients with renal stones
  - radiology
    - x-ray
      - may be useful in chronic gout
      - punched out lesions, areas of sclerosis, and in the latter stages, tophi, may be seen
      - first lesions usually occur in and around the first MTP joint
    - ultrasound, dual-energy CT and MRI
      - can identify urate deposition, structural joint damage and joint inflammation in gout
  - fasting glucose and lipids to rule out hyperglycaemia and hyperlipidaemia as gout is commonly associated with metabolic syndrome
- management
  - general
    - ice pack and rest may be useful
    - elevation of the joint, avoidance of trauma
    - pharmacological options include:
      - NSAIDs
        - first line treatment
        - the sooner they are started, the more rapid the response
        - there is no evidence to support any particular NSAID over any other
        - for patients with high risk of gastrointestinal adverse events, a gastroprotective agent should be used, or colchicine as an alternative
        - dose should be tailored to age, comorbidity and interactions, aiming for the highest tolerable licensed dose for the shortest possible time (in view of cardiovascular risk)

                - colchicine
                    - BNF recommends 500 micrograms 2-4 times/day until symptoms relieved (maximum 6mg per course and not repeated within 3 days)
                    - maximum dose is often limited by toxicity symptoms (nausea, vomiting, diarrhoea)
                    - particularly appropriate for patients who cannot tolerate NSAIDs, those on heart failure and those on anticoagulants
                    - can be effective at lower doses, should be titrated to maximum tolerable dose
                - corticosteroids
                    - can be given orally, IM, IV or intra-articularly
                    - useful when NSAIDs and colchicine are contraindicated
                    - short courses of low doses recommended (e.g. 15mg prednisolone/day or less)
                - other analgesics
            - choice for patient depends on:
                - contraindications
                - gap between onset of symptoms and start of treatment
                - risk vs benefit
        - discussion of lifestyle issues such as weight loss, exercise, diet, alcohol consumption and fluid intake
    - canakinumab
        - monoclonal antibody that may be used for patients whose condition has not responded adequately to treatment with NSAIDs or colchicine or who are intolerant of them
- if no improvement after 2-3 days of treatment:
    - review diagnosis
    - check medicine compliance
    - increase doses to the maximum
    - canakinumab
    - if still not improving, consider combining treatments or specialist advice
- prophylactic treatment between attacks
    - lifestyle modification
        - moderation with alcohol
            - keep to recommended limits
            - at least 3 alcohol-free days/week
            - beer and spirits should be avoided
        - avoid dehydration
        - reduction of purine based foods
            - reduction of meat or seafood
                - highest purine levels are in heart, herring, sardines and muscles

                - other foods include liver, kidneys, yeast extracts, oatmeal
                - soya foods are also high in purines but are less likely than meat or seafood to lead to gout
                - no evidence to support reduction in purine rich vegetables such as peas, beans, mushrooms or cauliflower
                - the quantity of purine rich food consumed is more important than the absolute purine content in each food
            - soft drinks containing fructose should be avoided
                - can affect levels of purine
            - weight reduction
            - regular exercise
            - smoking cessation
        - manage risk factors
            - drugs causing hyperuricaemia:
                - thiazides and loop diuretics
                - low dose salicylates
                - chemotherapy
            - hypertension
            - impaired renal function
            - hyperlipidaemia
            - vascular disease
            - myeloproliferative disease
        - prophylactic drugs
            - allopurinol
                - xanthine-oxidase inhibitor
                - normally lifelong and requires regular monitoring
                - may cause acute attacks of gout just after initiating treatment and for some weeks afterwards
                - should never be started during an acute attack (wait 1-2 weeks after resolution)
                - dose is titrated upwards as tolerated
            - uricosuric drugs
                - to increase excretion of uric acid in the urine
            - uric acid lowering therapy should be offered to patients with:
                - two or more attacks within a year
                - tophi
                - renal insufficiency
                - uric acid stones and gout
                - need for continuing diuretic therapy
- complications
    - renal disease
        - chronic urate nephropathy from deposition of crystals in the interstitium of medulla and pyramids causing inflammation and fibrosis

                - increased risk of urate and oxalate kidney stones
            - severe degenerative arthritis
            - secondary infections
            - recurrent painful episodes
            - carpal tunnel syndrome (rare)
            - nerve or spinal cord impingement
        - prognosis
            - first acute attacks usually resolve within 3-10 days
            - attacks recur in around 60% of people within a year
- pseudogout
    - background
        - inflammation of the joints caused by the deposition of calcium pyrophosphate (CPP) crystals in articular and periarticular tissues
            - CPP deposition also causes asymptomatic disease and chronic inflammatory disease
            - asymptomatic disease is common in the elderly with 50% showing radiographic changes
        - gender distribution is equal
    - precipitants
        - dehydration
        - intercurrent illness
        - hyperparathyroidism
        - long term use of steroids
        - hypothyroidism
        - any cause of arthritis
        - haemochromatosis
        - Wilson's disease
        - acromegaly
        - dialysis
        - surgery or trauma
        - hypomagnesaemia
    - presentation
        - often asymptomatic
        - acute CPP crystal arthritis
            - causes an acute monoarticular or oligoarticular arthritis
            - most often affects the knees
                - wrists, shoulders, ankles, hands and feet are also affected
                - almost any joint can be affected
            - presentation is similar to acute gout but normally milder
                - acute joint pain and swelling
            - affected joints are acutely inflamed with swelling, effusion, warmth, tenderness and pain on movement
                - may be associated with fever and raised WCC
        - chronic CPP crystal arthritis
            - destructive changes like OA but more severe
            - may progress to cause a destructive arthropathy producing a neuropathic joint

        - most often affects knees, wrists, shoulders and hips
    - differential
        - acute gout
        - OA
        - septic arthritis
        - osteoarthritis
    - investigations
        - joint x-rays
            - linear opacification of articular cartilage
        - ultrasound may be useful
        - aspiration of the joint fluid
            - raised WCC, predominantly neutrophils
            - glucose levels usually normal
            - intracellular and extracellular weakly positive birefringent crystals
                - intracellular crystals are pathognomic for acute pseudogout
            - joint fluid often looks purulent and septic arthritis must be excluded
    - management
        - no specific treatments for the elimination of CPP crystals from the body
        - treatment is symptomatic
        - most widely used treatments are:
            - ice, cool packs, temporary rest
            - aspiration of the joint
            - NSAIDs
            - intra-articular steroid injections
            - systemic steroids
            - colchicine (alternative if NSAIDs or steroids are contraindicated)
        - treatment of chronic CPP arthropathy is generally NSAIDs and PPI cover
    - prognosis
        - acute attacks usually resolve within 10 days
        - some patients develop progressive joint damage with functional limitation
        - prognosis depends on any underlying cause

MuC3 septic arthritis

- background
    - can destroy a joint in days
    - knee is most commonly involved in adults, hip in children
    - Staphylococcus aureus is the most common pathogen involved
        - mainly methicillin-sensitive strains
    - disseminated gonococcal arthritis is becoming more common among the elderly with multiple morbidities and in the immunosuppressed
        - incidence is 2.8 per 100,000 person years
        - usually presents with fever, arthralgia, multiple skin lesions and tenosynovitis of the hand joints, knees, wrists, ankles and elbows
            - may also present as an isolated monoarthritis
    - infection of the sternoclavicular and sacroiliac joints is commonly caused by Group B Strep
    - Lyme disease may cause swelling disproportionate to the level of pain

    - incidence of prosthetic joint infection ranges from 1.5-2.5% for primary intervention to up to 20% for revision procedures
- risk factors
    - increasing age
    - diabetes
    - prior joint damage
        - rheumatoid arthritis
        - gout
        - connective tissue disorders
    - joint surgery
    - hip or knee prosthesis
    - skin infection in combination with joint prosthesis
    - immunodeficiency
- presentation
    - single swollen joint with pain on active or passive movement
        - may present as polyarticular arthritis in a minority
    - majority of cases have fever and rigors but their absence does not exclude the diagnosis
        - bacteraemia is common
    - patients with septic arthritis of the sternoclavicular, acromioclavicular, sternocostal or manubrosternal joints may present with chest wall pain
    - infection of the sacroiliac joint may present as buttock, hip or anterior thigh pain
    - other associated conditions include recent steroid injection, sexually transmitted disease and IV drug use
- children
    - can easily be missed in children
    - localising signs may be absent
    - can be confused with more common conditions such as transient synovitis or trauma
    - patients usually present with fever, joint pain and/or unwillingness to move the affected joint
- signs
    - swollen, warm, tender joint
    - exquisitely painful on movement
    - may have obvious effusion
    - signs may be less obvious or poorly localised in the elderly, immunocompromised, drug abusers and in infections of the spine, hip and shoulder joints
    - viral arthritis (e.g. rubella, parvovirus, hep C, HIV) often presents with symmetrical involvement of smaller joints such as the hands, accompanied by a rash
    - tuberculous arthritis may be associated with a joint that feels ‘boggy’ on palpation due to granulomatous invovlement
    - infection of a prosthetic joint may show few signs until a drainage sinus develops
        - occasionally, an abscess around the joint or loosening of the implant is indicated by pain
        - may have a prolonged low-grade course with gradually increasing pain
- differential diagnosis
    - primary rheumatological disorders
    - drug-induced arthritis
    - reactive arthritis

    - Lyme disease
    - viral arthritis
    - infective endocarditis
- investigations
    - bloods
        - FBC (elevated WCC)
        - CRP/ESR (useful for monitoring response to therapy)
    - synovial fluids examination
        - for leucocyte count, Gram stain, polarising microscopy and culture
        - WCC is not diagnostic, culture is the only reliable method of evaluating a potentially infected joint
            - treatment should not be delayed for culture results
        - fluid can be injected directly into blood culture bottles to maximise the chance of growing organisms
        - aspiration of a prosthetic joint should only be attempted by an orthopaedic specialist
    - synovial tissue culture
        - where fungal or mycobacterial infection is suspected
    - blood cultures
        - at least two should be taken to exclude bacteraemia
    - cultures for gonococcal infection
        - rectal, cervical, urethral, pharyngeal
    - PCR
        - may be considered to detect bacterial DNA but cannot distinguish between live and dead organisms
    - Lyme disease tests if indicated
    - immunology
        - if suspicion of rheumatological disorders or vasculitides
- imaging
    - x-ray
        - limited value
        - may be normal in first few days
        - may show underlying osteomyelitis or periarticular osteomyelitis
        - fat pad displacement around the affected joint or joint space widening due to localised oedema and effusion
        - in later stages, diffuse joint space narrowing due to cartilage destruction
        - linear deposition of calcium pyrophosphate suggesting pseudogout
        - may show transcortical sinus tracts and new subperiosteal bone growth in prosthetic joint septic arthritis
    - ultrasound
        - less sensitive than MRI/CT
        - can be helpful to show intra-and extra-articular abnormalities and detect early effusion
        - can guide joint aspiration and drainage procedures
    - CT and MRI
        - most sensitive for diagnosing periarticular abscesses, joint effusions and osteomyelitis
        - usually reserved for diagnostically difficult cases

- management
    - surgical drainage and lavage of the joint
    - antibiotics
        - should be started empirically before the culture results are known
        - Gram stain results, age and sexual activity should guide initial antibiotic choice
            - should cover Staph aureus and Strep as a minimum
        - given IV initially
        - examples of treatments include:
            - specialist microbiology advice if there is a prosthetic joint
            - flucloxacillin for 4-6 weeks or longer (clindamycin as alternative)
            - if MRSA suspected, vancomycin or teicoplanin for at least 4-6 weeks
            - if gonococcal arthritis or Gram negative infection suspected, cefotaxime
        - IV treatment is often for 3-4 weeks
    - joint drainage
        - repeated percutaneous aspiration may be required if not responding to therapy
        - difficult to access joints may require ultrasound guidance or open arthrotomy
    - splinting
        - should be in the position of function
            - knees in extension
            - elbows at 90 degrees
            - wrists in neutral to slight extension
            - hips in balanced suspension in neutral rotation
        - immediate mobilisation once infection is under control promotes healing of the articular cartilage and prevents contractures
- prognosis
    - mortality rates of bacterial arthritis range from 10-20% depending on presence of comorbidities
    - factors associated with death include age 65 years or older, and infection in the shoulder, elbow or at multiple sites
    - patients with Staph aureus septic arthritis regain 45-50% of baseline joint function
    - morbidity occurs in a third of patients with bacterial arthritis (usually older patients, those with pre-existing joint disease and those with synthetic intra-articular material
        - includes amputation, arthrodesis, prosthetic surgery, severe functional deterioration

MuC4 limb pain and swelling: bursitis and tendonitis in the upper and lower limb including ruptured biceps, Achilles tendonitis, plantar fasciitis, metatarsalgia, carpal tunnel and other entrapment neuropathies plus sinister causes including bone tumour, stress fracture

- bursitis
    - background
        - a bursa is a small sac of fibrous tissue with a thin synovial lining filled with fluid

        - numerous bursae are found in the body, around joints or where ligaments and tendons pass over bones
        - they can also form in other places as a response to unusual pressure or friction
        - they generally help to reduce friction and allow maximal range of movement around joints
        - bursitis is inflammation within a bursa, which leads to an increase in synovial fluid production and swelling of the bursa
    - olecranon bursitis
        - background
            - the bursa lies over the ulna at the posterior tip of the elbow
            - near the surface and frequently subject to trauma
            - often caused by constant irritation whilst leaning on a table but can be caused by repetitive elbow movement or isolated trauma
            - can be infected or non-infected
            - more common in:
                - men
                - young to middle age
                - occupations causing regular trauma to or pressure on the elbow (e.g. gardeners, mechanics, carpet layers, students)
                - sportspeople with repetitive elbow flexion or overhead throwing or who are at risk of frequent falls onto the elbow
                - most cases of septic bursitis are caused by Staph aureus, with strep the next most common cause
        - history
            - focal swelling over the posterior tip of the elbow, may or may not be painless
                - pain tends to lessen with chronicity
                - pain is often exacerbated by pressure, such as leaning on a table
            - acute onset without trauma is suggestive of infection
        - examination
            - clearly demarcated swelling in the region of the posterior elbow tip
                - often has the appearance of a ‘goose egg’
            - area may be tender to palpation, with redness and warmth, particularly if infection present
            - there may be skin contusion or abrasion if there was a recent injury
            - range of joint movement is usually normal but may be limited at end of flexion due to pain from compression of the bursa
                - unusual restriction of active or passive movement and a history of trauma raises the suspicion of an olecranon process fracture
            - signs suggestive of infection:
                - pain
                - bursa is hot to touch
                - increasing swelling
                - surrounding cellulitis

        - broken skin over the bursa
        - fever (with advanced infection)
- investigations
    - usually a clinical diagnosis – other tests may be considered in diagnostic uncertainty
    - bloods
        - raised WCC suggestive of infection
        - uric acid
        - rheumatoid factor
        - ESR/CRP
    - imaging
        - x-ray
            - if there is a history of significant trauma to exclude olecranon process fracture
        - ultrasound
            - may detect effusions, synovial proliferation, calcifications, loose bodies, rheumatoid nodules, gouty tophi, septic processes
        - MRI may be considered
    - aspiration
        - useful to check for septic process and for relief of symptoms
        - an 18 or 20 gauge needle is inserted directly into the bursa with the elbow flexed, from a lateral approach to avoid the ulnar nerve
        - fluid should be sent for microscopy and culture
- associated diseases
    - diabetes
    - rheumatoid arthritis
    - gout
    - HIV
    - alcoholism
    - CKD (due to uraemia or repeated microtrauma during dialysis)
    - immunosuppressive treatment
- management
    - non-septic bursitis
        - reduced activity
        - ice
        - compression bandage may ease discomfort
        - protect elbow from trauma
        - paracetamol or NSAIDs for analgesia
        - other treatment options include:
            - aspiration of bursa
            - corticosteroid injection into the bursa
            - if an olecranon spur contributes to recurrence, surgical removal of the spur is helpful
    - septic bursitis

        - treat empirically with antibiotics whilst awaiting culture results (usually flucloxacillin or erythromycin)
        - surgical drainage or bursectomy may be required in some cases
    - complications
        - secondary infection after aspiration or steroid injection
        - sepsis and osteomyelitis in severe septic bursitis
        - fistulae following surgical drainage or spontaneous rupture
        - persistent pain and decreased function in ongoing/recurrent cases
    - prognosis
        - most patients recover completely with no complications
        - recurrence is more likely when it is caused by repeated minor trauma
- prepatellar bursitis
    - background
        - there are four bursae around the knee joint
        - all can be affected by bursitis but the prepatellar bursa is most commonly involved
        - the prepatellar bursa is located superficially on the anterior aspect of the knee between the skin and the patella
        - the bursa does not communicate with the knee joint
        - can affect all age groups
    - causes
        - acute trauma
            - fall/direct blow to the knee
        - recurrent minor injury
            - long periods of time kneeling forwards and putting pressure on the patella
        - infection
            - pyogenic prepatellar bursitis is common in children and may be mistaken for septic arthritis of the knee; it is usually caused by Staph aureus and there is usually a history of a break in the skin prior to onset
        - co-existing inflammatory disease
        - crystal-depositing condition
    - symptoms
        - knee pain, swelling and redness
        - difficulty kneeling and walking
        - fever
    - signs
        - tenderness and swelling superficial to the patella
        - erythema and localised warmth of the skin over the patella
        - reduced knee movement
        - in septic bursitis, fever, tachycardia, signs of systemic upset
    - key points from history
        - occupation and whether it involves excessive kneeling
        - history of fall or acute knee injury

- history of repetitive motion involving the knee
- any recent steroid treatment
- whether the patient is immunocompromised
- any history of crystal arthropathy or inflammatory disease

- differential
  - patellofemoral pain syndrome
  - septic arthritis of the knee
  - cellulitis
  - knee joint effusion secondary to trauma
  - infrapatellar bursitis
- investigations
  - aspiration of the bursa
    - to differentiate septic and non-septic bursitis
  - imaging
    - not usually required, may be considered if fracture or dislocation is possible
    - MRI, CT and US may also be considered
- management
  - non-septic
    - rest
    - ice
    - analgesia (paracetamol, ibuprofen)
    - patient education
    - thick foam cushion or knee pads to help prevent recurrence; occupational therapy may be helpful
    - physiotherapy if there is reduced range of movement; a stick or cane may be needed
    - surgical treatment may be required if chronic or recurrent (arthroscopic or open bursectomy)
  - septic bursitis
    - aspiration
    - antibiotics
    - incision and drainage if symptoms do not improve within 36-48 hours of antibiotic treatment
- prognosis
  - can lead to pain and reduced function of knee joint
  - generally very good prognosis with appropriate treatment

- tendonitis
  - background
    - inflammation or irritation of a tendon
    - commonly occurs in athletes or middle aged or older people
    - associated with excessive strain, repetitive trauma or unaccustomed exercise
    - fluoroquinolone antibiotics and statins may increase risk
    - clinical features are pain with movement, particularly at the insertion site, and mild swelling
    - types include:

- lateral epicondylitis
    - overuse syndrome affecting the forearm, wrist and digit extensors/supinators
    - tender to palpation over the lateral epicondyle, pain with forced extension and supination of the forearm
    - counterforce brace may be used for immobilisation
- medial epicondylitis
    - overuse syndrome affecting the wrist and digit flexors
    - tenderness to palpation over medial epicondyle, pain with forced flexion and pronation of forearm/wrist
    - ulnar neuropathy may develop
    - counterforce brace may be used for immobilisation
- patellar tendonitis
    - seen in runners, basketball and volleyball players, high jumpers
    - pain is referred to the area of the patellar tendon
    - pain worse on going from sitting to standing and running (especially uphill)
    - quadriceps strengthening exercises are used as part of management
- De Quervain tenosynovitis
    - tenosynovitis of abductor pollicis longus and extensor pollicis brevis
    - more common in middle age and women
    - associated with rheumatoid arthritis and SLE and overuse of the thumb (e.g. lifting infants, secretarial/nursing work or heavy texting)
    - pain along radial aspect of wrist which may radiate to the thumb or forearm
    - there is painful abduction of the thumb and decreased grip strength
    - positive Eichoff test (patient grasps thumb in palm of hand and ulnar deviates the hand and thumb; stretches the tendons over the radial styloid, producing sharp pain
    - positive Finkelstein's test
        - practitioner grabs thumb and sharply ulnar deviates, causing sharp pain
    - negative Phalen's and Tinel test
    - x-rays are non-diagnostic; US is diagnostic
    - management is splinting of the thumb and wrist (removed briefly daily to perform range of motion exercises), NSAIDs for 10-14 days, for persistent cases – steroid injection or surgical decompression
- rotator cuff tendinitis
- bicipital tendinopathy
    - occurs in the long head of biceps
    - tends to occur with repetitive overhead activity

        - diagnosis is clinical
        - initial general management as for other tendinopathies, with orthopaedic referral if not improving after 2 months
    - popliteus tendinopathy
    - iliotibial band syndrome
        - caused by excessive friction between the iliotibial band and the lateral femoral condyle
        - presents with activity related knee pain
        - clinical diagnosis
            - tenderness over the lateral femoral condyle made worse with a single leg squat
            - tightness of the iliotibial band (Ober's test)
                - patient is lateral with symptomatic side up and knee flexed to 90 degrees; the hip is brought from flexion and abduction into extension and adduction
        - treatment is nonoperative with rest, NSAIDs and stretching of the iliotibial band, quadriceps and gluteal muscles; surgical release is rarely required for chronic/refractory cases
    - medial tibial stress syndrome ('shin splints')
        - overuse injury or repetitive load injury of the shin area leading to persistent dull anterior leg pain
        - diagnosis is clinical with tenderness along the posteromedial distal tibia made worse with plantar flexion; x-rays or bone scans may be done to rule out stress fractures
        - treatment is generally nonoperative with NSAIDs, rest and activity modification
- imaging
    - US
        - may show tendon disruption, increased blood flow, hypoechoic areas or tendon thickening
    - x-ray
        - if concerns about fracture, foreign body or avulsion
    - MRI
- management
    - specific to type of tendonitis
    - general management
        - rest/load reduction
        - ice/heat
        - NSAIDs
    - consider physiotherapy and oral steroids

- tendon rupture
    - background
        - usually diagnosed by clinical assessment
        - x-rays or ultrasound may be used to establish or confirm diagnosis

                - MRI gives the most definitive information about the nature and extent of the rupture
            - uncommon, but can cause severe pain and lead to permanent disability if untreated
            - management may be surgical or non-surgical
            - patients over 60 are more at risk
            - tendon damage may be associated with quinolone use (e.g. ciprofloxacin, levofloxacin)
                - can occur within 48 hours of starting or several months after stopping
                - risk is increased by concomitant use of steroids
    - proximal biceps tendon rupture
        - background
            - nearly all biceps ruptures
            - usually transverse and either within the shoulder joint or within the proximal part of the intertubercular groove
            - prognosis is good for both surgical repair and conservative management
        - presentation
            - the biceps muscle bunches up in the distal arm, causing the characteristic 'Popeye muscle' appearance
            - minimal loss of function
        - management
            - normally conservative
            - most become asymptomatic after 4-6 weeks
            - they may benefit from NSAIDs and physiotherapy
            - no generally agreed guidelines for the role of surgical repair
                - tenodesis and proximal subacromial decompression or distal reattachment may be required for young or athletic people or people who require maximum supination strength
    - distal biceps tendon rupture
        - background
            - usually caused by a single traumatic event involving flexion against resistance with the elbow at a right angle
            - incidence 1.2 per 100,000 per year
            - most often occurs in middle aged active males
        - presentation
            - a sudden sharp tearing sensation followed by a painful swollen elbow with weakness of flexion and supination
            - in a partial rupture, the biceps tendon will still be palpable in the antecubital fossa
        - management
            - conservative treatment results in persistent elbow weakness, especially supination
                - patients may experience prolonged pain

        - surgery must be performed early in order to avoid scarring of the biceps; with delayed treatment the biceps may be attached to the brachialis
    - complications
        - rupture may lead to biceps tendonitis and median nerve compression
- patella tendon rupture
    - background
        - usually unilateral and due to a sports injury in patients younger than 40
        - bilateral ruptures can occur in more minor traumas in patients with systemic conditions such as inflammatory disease, diabetes, CKD
    - presentation
        - immediate onset of pain with a tearing sensation
        - diffuse tender swelling with bruising in the anterior knee
        - a defect at the level of the rupture may be palpable
        - active extension may be completely lost and the patient unable to maintain the passively extended knee against gravity
    - management
        - conservative management has a very limited role
        - may be indicated for rare cases of partial patella tendon tear
            - cast or brace immobilisation in full extension followed by physiotherapy
        - immediate surgical repair of the ruptured tendon is recommended for optimal return of function
            - outcome is closely related to time from injury to repair
            - if it is repaired immediately, most patients experience nearly full return of knee motion
- quadriceps tendon rupture
    - background
        - relatively infrequent, usually in patients over 40
        - spontaneous rupture may occur; the most common mechanism is a fall
    - presentation
        - typically acute knee pain, swelling and functional loss following a stumble, fall or giving way of the knee
        - there may be no history of prior knee pain
        - suprapatellar swelling, bruising and tenderness are present
        - there is variable loss of knee extension
    - management
        - early surgical repair gives the best results for complete ruptures
        - partial tears can be treated conservatively with rest, analgesia and physiotherapy
- posterior tibial tendon rupture
    - background

- the posterior tibial tendon maintains the arch of the foot and rupture is one of the most common causes of acquired flat foot in adults
- the foot may become so deformed that severe ankle arthritis develops

- presentation
  - pain frequently begins just behind the medial malleolus
  - the foot rolls inwards and becomes flat
- management
  - surgery indicated for greater degrees of foot deformity
    - tenosynovectomy, lateral column lengthening or arthrodesis
  - arch supports and heel cups are usually ineffective in relieving symptoms
    - the objective is to reduce excessive midfoot motion using total contact orthosis supporting the longitudinal arch, and a medial heel wedge

- peroneal tendon rupture
  - background
    - most are tears of the peroneus brevis, usually occurring as the result of a lateral ankle sprain
    - tears have been linked to ballet dancing, skiing, football, tennis, running, basketball and ice skating
    - the longer the injury takes to heal, the greater the suspicion of a tendon rupture
    - many cases are too small to find with any test other than exploratory surgery
  - management
    - conservative treatment with immobilisation followed by mobilisation and physiotherapy can be tried but has a relatively high recurrence rate
    - acute peroneus longus tears more commonly occur at the level of the cuboid tunnel and may initially be managed non-operatively
      - may require debridement and tenodesis
    - complete ruptures of both peronei are rare and require reconstructive surgery if there is significant defect
- hand flexor tendon rupture
  - background
    - most often, the flexor tendons are damaged by a cut, which may also damage adjacent nerves
    - closed flexor tendon ruptures due to trauma without an external wound are rare
    - sports injuries are also common causes, usually in football, wrestling or rugby
    - people with rheumatoid arthritis may have spontaneous rupture of the hand tendons
  - presentation
    - tenderness over the flexor aspect of the finger

- the patient is unable to flex one or more joints of the finger and attempting to do so causes pain
        - management
            - surgical repair is required and should be done as soon as possible
    - hand extensor tendon rupture
        - background
            - extensor tendons are easily injured, even by a minor cut
            - jamming a finger may cause the tendon to tear from the attachment to the bone
        - presentation
            - hand extensor tendon rupture can cause a mallet finger (fixed flexion of distal interphalangeal joint) or boutonnière deformity (fixed flexion at the middle interphalangeal joint)
            - lacerations on the back of the hand that go through the extensor tendons can cause inability to extend the finger at the MCP joint
        - management
            - partial injuries are treated conservatively, with a splint
            - complete rupture is treated by operative repair
            - surgery to free scar tissue may be needed if there is significant loss of finger movement
- Achilles tendonitis and rupture
    - Achilles tendinopathy
        - background
            - chronic overuse injury of the Achilles tendon
            - can affect anyone but most common in active people, especially participants in sports with running and jumping
            - the tendon begins near the mid calf and inserts into the posterior of the calcaneus, connecting the gastrocnemius and soleus muscles to the ankle
            - it is surrounded by a connective tissue sheath (paratenon) rather than a true synovial sheath
                - the paratenon is very vascular and highly innervated and stretches with movement, allowing maximum gliding action
            - near the insertion of the tendon are two bursae (subcutaneous calcaneal and subtendinous calcaneal) and Kager's fat pad – all can be a source of heel pain
            - Achilles tendinopathy generally occurs in the midsubstance of the tendon, less commonly at the insertion (20-25%)
                - the cause is not fully understood but is a maladaptive response to increased loading leading to thickening and stiffness of the tendon, then disrepair and degenerative changes
            - typically affects athletes
                - long distance runners have a lifetime risk of 52%
            - annual incidence of 2.4 per 1000 adults registered with a GP
            - no gender difference
        - risk factors

        - running, jumping, dancing and tennis
        - rapid increase in the amount of time spent on the activity
        - change in footwear or training surface
        - poor running technique such as excessive pronation
        - poorly fitting footwear
        - family history (increases risk x5)
        - hypertension
        - diabetes
        - familial hypercholesterolaemia may present with Achilles' tendon pain
        - inflammatory and autoimmune arthritis should be considered with insertional tendinopathy
        - quinolone antibiotics can cause inflammation of the tendon and predisposition to rupture
- presentation
    - gradual onset of pain and stiffness over the tendon
        - may improve with heat or walking and worsen with strenuous activity
    - tenderness of the tendon on palpation
        - mid substance tendinopathy is tender between 2-6cm above the calcaneal insertion
        - insertional tendinopathy is tender at the insertion
    - there may be crepitus and swelling
    - pain on active movement of the ankle joint
    - rupture of the tendon should always be excluded with Thompson's test
- investigations
    - US or MRI may be necessary if the diagnosis is unclear and to differentiate between tendinopathy and partial thickness tears
- management
    - immediate relief:
        - rest
        - ice
        - analgesics including NSAIDs
        - heel lifts – orthotic devices, used on both sides to prevent a gait imbalance
    - gentle stretching of the front and back of the lower leg
    - patient will need to alter activities, at least in the short term – may benefit from advice from a physiotherapist
    - if no improvement after 10 days of rest and gentle stretching, eccentric heel drop exercises are the best treatment
        - pushing up on the toes of both feet then lowering over the edge of a step on the affected foot
        - two exercises, one done with the knee straight, one in flexion
            - 15 repeats of each should be done twice a day
        - may be painful

        - should be done daily for 3 months, with increasing load, and may need to be continued for much longer
      - steroid injections into or around the tendon are not approved by NICE and may cause tendon atrophy and rupture
      - combining eccentric loading with extracorporeal shock wave therapy is effective
      - casting can be an option for resistance tendinopathy
      - surgery is sometimes used, usually as a last resort
    - prognosis
      - recovery takes weeks to months
      - 85% have completely normal function with no symptoms 8 years after initial injury
- Achilles tendon rupture
  - background
    - can occur at any age but most common in recreational athletes aged 30-50 years
    - commonly seen in football, running, basketball, diving, tennis and other sports that require forceful push off with the foot
    - incidence is 7 injuries per 100,000 of the general population
    - other causes are:
      - injury, such as a fall, with forced dorsiflexion of the foot (sudden stretch on tendon)
      - deep lacerations over the site of the tendon
    - diagnosis is not always obvious and can be easily missed
      - it is important to look actively for evidence of tendon rupture, do Thompson's test and refer if rupture suspected
  - risk factors
    - increasing age
    - chronic/recurrent Achilles tendinopathy
    - steroids
      - systemic prolonged or high dose corticosteroids or Cushing's syndrome
      - previous steroid injections into or around the Achilles tendon
    - systemic conditions
      - gout, rheumatoid arthritis, SLE
    - quinolone antibiotics
      - triples risk, increased further with concomitant steroid use
- history
  - acute onset of pain in the tendon, felt as a sudden sharp pain initially
    - sometimes described as the feeling of being hit in the back of the leg
    - a snap may be heard as the tendon ruptures
    - the pain then settles into a dull ache
  - there may be a history of less intense pain for several days before rupture
  - patients may notice an inability to stand on tiptoe and altered gait (inability to push off with the affected foot)
- examination

        - observe gait
        - there is usually localised swelling
        - there may be a palpable defect in the Achilles tendon if rupture is complete rather than partial
            - the tendon defect may be masked by bruising
        - active plantar flexion is weak or absent
            - some active flexion may be possible through the action of other muscles
        - Thompson's test
            - performed with the patient lying prone with the knee passively flexed
            - absence of normal plantar flexion on squeezing the calf muscles indicates a complete rupture
            - compare with the other leg
    - differential
        - Achilles tendonitis
        - retrocalcaneal bursitis
        - plantaris muscle injury
        - posterior ankle impingement
        - sural nerve entrapment
        - other ankle injuries
        - ankle osteoarthritis
        - ruptured Baker's cyst
        - deep vein thrombosis
    - investigations
        - ultrasound or MRI may help if the diagnosis is unclear
    - management
        - non weight bearing as soon as rupture suspected
        - urgent referral to orthopaedic specialist
        - treatment options
            - open surgical treatment significantly reduces the risk of rerupture compared with non surgical treatment but has significantly higher risk of complications such as wound infection
                - percutaneous surgery may decrease risk of wound infection
                - surgery may be recommended for competitive athletes and others with high physical activity, if there has been a delay in diagnosis and treatment and for those with recurrent rupture
            - conservative management is an option, especially for older and less athletic patients
                - rest, pain control, walking boot from two weeks and weight bearing as tolerated from 4-6 weeks
                - physiotherapy to guide controlled early motion
            - use of brace rather than plaster cast may reduce complications
    - complications
        - Achilles tendon contracture and/or scarring may occur from excessive immobility

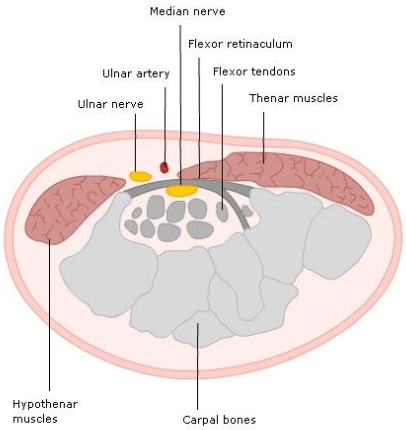
        - re-rupture
        - DVT has a higher incidence after Achilles tendon rupture
    - prognosis
        - prognosis is good but the slight loss of function may be significant for competitive athletes
        - athletes may be unable to resume full activities for one year
- plantar fasciitis
    - background
        - widely used as a term for undersurface heel pain
        - common problem
        - no male/female difference
        - 5-10% of running injuries are plantar fasciitis
        - pathology is not the result of excessive inflammation
        - pathological changes are degenerative but partially reversible, probably due to repetitive trauma
        - the plantar fascia is a thick fibrous band of connective tissue
            - its origin is the medial plantar tubercle of the calcaneum
            - runs along the sole of the foot like a fan, attached at the other end to the base of each toe
            - functions during running and walking:
                - stabilises the metatarsal joints during impact with the ground
                - acts as a shock absorber for the entire leg
                - forms the longitudinal arch of the foot and helps to lift the arch to prepare it for the 'take off' phase of the gait cycle
    - pathophysiology
        - the degree of stress makes the fascia susceptible to injury
        - a force equal to around three times the body's weight passes through the foot with each step
            - happens around 90 times a minute when running
        - plantar fasciitis is thought to be a traction and overuse injury
            - damage to the fascia is normally in the form of micro tears, it is a degenerative rather than inflammatory process
        - damage tends to occur near the heel, where stress on the fibres is greatest and the fascia is thinnest
        - plantar fasciitis is often associated with calcaneal spurs – depositions of calcium where the fascia suffers most damage
            - spurs are most commonly on the medial side at the origin of the fascia from the calcaneum
            - they are the result of the process of plantar fasciitis and not the cause of the pain
        - it can present bilaterally
    - risk factors
        - participants in sport that involve some degree of running and jumping
        - non-athletic people who spend much of the day on their feet
        - may occur in someone who suddenly becomes more active after a period of relative inactivity

        - increased risk from running on hard ground and increased hill training
        - worn-out trainers increase risk as they lose shock absorption properties
        - obesity – due to increased stress through fascia
        - other mechanical factors such as flat feet (pes planus) and high arch (pes cavus)
        - pregnancy – temporary physiological gain in weight and relaxation of ligaments due to hormones which predisposes to flat feet
        - may be an association with HLA B27 associated spondyloarthropathies
    - history
        - main complaint is heel/planar pain
            - most often 1-2cm distal to the medial calcaneal tuberosity
        - onset of symptoms and any precipitating, aggravating or relieving factors
            - often most severe during first few steps after prolonged inactivity such as sleeping or sitting
            - sitting with the foot elevated usually relieves pain
            - for people on their feet all day, pain is worst at the end of the day
            - walking barefoot, on toes or upstairs can precipitate pain
        - running, jogging or other sports
        - footwear and when it was last replaced
        - previous trauma to the foot
    - examination
        - look
            - obvious deformities, skin changes or congenital conditions
            - pes planus or pes cavus
        - there is often tightness of the Achilles tendon and ankle dorsiflexion may be limited
        - palpation of plantar surface over medial calcaneal tuberosity and along the course of the plantar fascia
            - pressing quite hard
            - reproduction of pain is the most important sign to confirm diagnosis
        - pain may be reproduced by asking the patient to stand on their toes or by passive dorsiflexion of the toes
        - palpate the back of the heel and ankle to exclude Achilles tendonitis
        - there should be little or no swelling
            - swelling may suggest subcalcaneal bursitis
        - referred pain from S1/A2 lesion should be excluded
            - perform straight leg raising test
            - check ankle tendon reflex (S1) and calf strength
                - ask patient to walk on toes or stand on one leg and raised the heel off the floor
        - exclude tarsal tunnel syndrome
            - the posterior tibial nerve passes under the flexor retinaculum between the medial malleolus and the calcaneum
            - percuss over the nerve below and posterior to the medial malleolus
                - can reproduce pain, numbness and burning on the medial side of the foot, ankle or calf if there is tarsal tunnel syndrome

        - press together the heads of the 2nd and 3rd metatarsals then the 3rd and 4th
            - reproduction of pain suggests Morton's neuroma with entrapment of the common digital nerve between the metatarsal heads
        - stress fracture of the calcaneum causes tenderness over the calcaneum rather than anterior to it
    - differential
        - Achilles tendonitis
        - subcalcaneal bursitis
        - S1 radiculopathy or referred pain
        - tarsal tunnel syndrome
        - Morton's neuroma
        - Sever's disease (children and adolescents)
        - stress fracture of calcaneum
        - rare causes:
            - fibrosarcoma
            - metastases
            - foreign body
            - Paget's disease of the bone
            - osteomyelitis
            - tuberculosis
            - gout
    - investigations
        - no role for bloods
        - weighing and measuring the patient can help to reinforce the need for weight loss if obese
        - x-ray
            - should not be routinely performed
            - may be indicated if another diagnosis suspected
            - lateral view may show soft tissue calcifications or a calcified spur on the anterior aspect of the calcaneus
            - may help to exclude a stress fracture although these are not always evident on x-ray
        - ultrasound
            - may show a thickened heel aponeurosis
        - bone scans and MRI
            - may be considered
    - management
        - no one treatment with strong evidence of efficacy
        - general advice
            - resting of the foot as much as possible
            - loss of weight if obese
            - correction of pes planus if present
            - advice to run on a softer surface
            - a laced sport shoe and regularly updated shoes
            - discuss training schedules with athletes with consideration of a switch to swimming, cycling, step machine or other low impact exercise

                - NSAIDs and ice
                - patients should not expect a significant improvement in symptoms before 6-8 weeks with a conservative management plan
            - physiotherapy and stretching
                - stretching exercises
                - deep massage of the sole of the foot to stretch the plantar fascia
            - orthotics, splinting and casting
                - a heel and arch support may help
                    - inserts should be worn in both shoes even if the pain is unilateral
                - night splints to keep the ankle dorsiflexed and toes extended to stretch the plantar fascia may help
            - corticosteroid injection
                - may provide some short term relief
            - other modalities
                - extra-corporeal shockwave therapy – limited evidence on efficacy but appears safe
                - Botox injections – may give some short term relief
                - autologous blood injection – lacking long term data, corticosteroid injections are probably superior
                - radiotherapy provides effective pain relief
                - surgery to release the plantar fascia from the bone may be considered for refractory symptoms
    - prognosis
        - pain may be longstanding and can last for years
        - generally resolves over time with minimally invasive management
        - 80% show spontaneous response within 12 months
    - prevention
        - regularly changing footwear used for running and walking
        - wearing shoes with good cushioning in the heel and good arch support
        - losing weight if overweight
        - avoid exercising on hard surface
        - regular stretching exercise for the plantar fascia and Achilles tendon
- metatarsalgia
    - pain across the metatarsal heads
    - often due to collapse of the transverse arch
    - a range of abnormalities including plantar plate tears may be visible on ultrasound
    - a metatarsal pad with often reform the arch and provide pain relief
        - a podiatrist may be able to help
    - surgical treatment may be required for severe rheumatoid arthritis changes
- nerve entrapment syndromes
    - background
        - a group of conditions in which peripheral nerves are damaged through compression or repeated trauma
        - the three most common are:
            - median nerve at the wrist (carpal tunnel syndrome)
            - ulnar nerve at the wrist (Guyon's canal) and elbow

            - radial nerve in the forearm
        - the pathological process and principles of treatment are similar for each
    - pathophysiology
        - caused by chronic injury to the nerve as it travels through a bony or ligamentous tunnel
            - repetitive compression, rubbing or sliding occurs
        - immobilisation with a splint can reduce this form of damage
        - entrapment neuropathies can also be caused by systemic disorders such as rheumatoid arthritis, pregnancy, acromegaly or hypothyroidism
        - repetitive injury can result in ischaemia, oedema and damage to the myelin sheath of the nerve
            - focal segmental demyelination at the site of compression is the key pathophysiological feature of all nerve entrapment syndromes
            - complete recovery of function after surgical decompression reflects remyelination of the affected nerve
            - incomplete recovery in more chronic or severe cases is due to Wallerian degeneration of the axons and permanent fibrotic changes in the neuromuscular junction which prevent reinnervation and restoration of function
    - clinical presentation
        - depend on the nerve involved
        - symptoms can be irritative (pain, paraesthesia) which affect sensory nerves and follow a temporal sequence
        - if not treated, ablative symptoms follow and may be irreversible
            - numbness – sensory nerves
            - weakness and atrophy – motor nerves
        - in some mixed sensory and motor nerves, for example the median nerve, signs of sympathetic dystrophy may occur in chronic cases:
            - dry, thin, hairless skin
            - ridged, thickened, crackly nails
            - recurrent skin ulcerations
    - management
        - initial management
            - analgesia
            - reducing the aggravating movement (splinting or change of activity)
        - surgical decompression may be required if this fails
    - carpal tunnel syndrome



https://www.rcemlearning.co.uk/modules/common-entrapment-syndromes/lessons/median-nerve-at-the-wrist-carpal-tunnel-syndrome/topic/anatomy-5/

- background
  - incidence 2.5 cases per 1000 people per year
  - compressive neuropathy of the median nerve at the wrist
  - more common in women and in middle age
- anatomy
  - the carpal tunnel is located at the base of the palm
    - bounded on 3 sides by carpal bones and anteriorly by the transverse carpal ligament (flexor retinaculum)
  - the median nerve runs inside the carpal tunnel with the flexor tendons and their synovial sheaths
  - any condition that causes an increase in swelling of the contents of the carpal tunnel can result in compression of the median nerve
- history
  - typically pins and needles or a burning sensation in the median distribution of the hand
  - generally worse and night and relieved by shaking the hand
  - normally affects at least two of thumb, index finger and middle finger on the palmar surface
  - symptoms affecting the ring and little fingers, wrist pain and radiation of pain proximal to the wrist may occur, but are uncommon
    - carpal tunnel syndrome is unlikely if there are no symptoms in the first three digits
  - symptoms are usually bilateral and progressive with an insidious onset
  - in the later stages, an aching sensation may radiate to the forearm and elbow
- risk factors
  - compression of tunnel walls
    - rheumatoid arthritis

        - trauma, e.g. Colles' fracture
        - acromegaly
        - subluxation of the wrist
    - internal tunnel compression
        - myxoedema
        - oedema after repetitive movements
        - fluid retention in pregnancy
        - chronic proliferative synovitis
    - changes in the median nerve
        - peripheral neuropathy
        - diabetes
- examination
    - weakness of thumb abduction is common
    - hyperflexion of the wrist for 60 seconds may elicit paraesthesia in the median nerve distribution (Phalen's sign)
    - loss of two point discrimination or abductor pollicis brevis atrophy
    - Tinel's sign
        - tapping the volar wrist over the median nerve may produce paraesthesia in the median distribution of the hand
- differential
    - tendonitis
    - tenosynovitis
    - compressive neuropathies of the nerve roots and brachial plexus
    - proximal median neuropathy
    - polyneuropathy
- investigations
    - diagnosis is usually clinical
    - ultrasound may be helpful
    - electromyographic and nerve conduction studies
        - most helpful for determining site and severity of compression
        - it is possible to have normal studies with carpal tunnel syndrome
- management
    - conservative
        - volar splint place in a neutral position
            - initial success rate up to 70%, falls over time
        - no benefit of ultrasound treatment
        - corticosteroids are beneficial for one month with unclear benefit after this
    - surgery
        - definitive therapy is surgical release of the transverse carpal ligament
        - relieves symptoms significantly better than splints
        - endoscopic surgery has more transient nerve problems, open surgery has more wound problems
        - long term success rate >75%

- prognosis
    - excellent with definitive surgery
    - more benign course during pregnancy with fewer cases requiring surgical treatment
    - risk factors for poorer than average prognosis include:
        - advanced disease
        - atypical symptoms (normal nerve conduction studies, symptoms in fifth digit)
        - longer symptom duration
        - older age
        - co-existing disease (diabetes, other peripheral neuropathy)
        - heavy manual occupation
    - some patients have residual fingertip numbness despite treatment

- ulnar nerve entrapment at wrist and elbow
    - background
        - second most common nerve entrapment neuropathy
        - can occur anywhere along its length but most common at the elbow
            - cubital tunnel and epicondylar groove are the most common sites
            - compression may be caused by a congenitally shallow epicondylar groove, post trauma, tumour, infection, arthritic spurs
        - damage to the motor component can cause loss of intrinsic muscle function in the hand
        - second most common area from compression is Guyon's canal
    - presentation (wrist)
        - patient may have motor, sensory or mixed symptoms depending on the site of compression
        - zone 1 – proximal to the bifurcation of the ulnar nerve
            - mixed loss
            - commonly caused by fractured hook of hamate or a ganglion
        - zone 2 – motor branch after bifurcation
            - loss of motor supply to muscles innervated by the ulnar nerve in the hand
            - fracture of the hook of hamate is the most common cause
        - zone 3 – sensory branch after bifurcation
            - sensory loss to the hypothenar eminence (little finger and lateral side of ring finger)
            - most common causes are aneurysm of the ulnar artery, thrombosis and synovial inflammation
    - presentation (elbow)
        - commonly due to mild repetitive injuries if traumatic cause

        - often repeated flexion and/or extension or resting on the elbow for a prolonged period of time
        - spontaneous subluxation of the ulnar nerve out of the cubital tunnel can occur, aggravating symptoms of entrapment
    - symptoms vary from mild transient paraesthesia to severe intrinsic muscle atrophy
    - pain may be in the elbow or wrist and may radiate to the hand or shoulder
    - in mixed involvement, numbness usually precedes motor loss
    - early muscle weakness can cause difficulty in opening bottles or doors or early fatigue in repetitive movements and may be painless
- examination
    - dorsal sensation to the ring and little finger is preserved in wrist entrapment, but not in elbow entrapment as the dorsal cutaneous branch of the ulnar nerve leaves the ulnar nerve about 9cm proximal to the wrist
    - intrinsic muscle function is tested by asking the patient to cross the index and middle fingers
    - Froment's sign
        - weakness of adductor pollicis is demonstrated by eliciting Froment's sign when grasping a piece of paper
        - the flexor pollicis longus causes the thumb to flex because adduction is insufficient
    - ulnar claw
        - a claw hand is produced by lesions below the mid forearm
            - in lesions above this, clawing does not occur because the long flexors are also denervated
- differential
    - cervical disc disease
    - brachial plexus abnormalities, thoracic outlet syndrome, Pancoast tumour
    - elbow abnormalities, epicondylitis
    - infections, tumours, diabetes, hypothyroidism, rheumatoid diseases, alcoholism
    - wrist fractures
    - ulnar artery aneurysms or thrombosis at the wrist
- investigations
    - imaging
        - x-rays of the elbow and wrist are required because entrapment may occur at more than one level
        - elbow x-rays may show abnormal anatomy such as a valgus deformity, bone spurs, bone fragments,

shallow olecranon groove, osteochondromas, destructive lesions (e.g. tumours, infections, abnormal calcifications)
                - wrist x-rays may show fractures of the hook of hamate, dislocations of the wrist bones, soft tissue masses and calcifications
                - MRI is not normally needed unless visualisation of soft tissue or nerves is needed
            - diagnostic procedures
                - electromyography tests and nerve conduction studies
                    - to confirm area of entrapment, document extent of pathology and detect or rule out possibility of double crush syndrome (compression in more than one place)
                - Tinel's and Phalen's tests can also be used
        - management
            - conservative
                - most successful when paraesthesia is transient and caused by malposition of the elbow or blunt trauma
                - NSAIDs to relieve nerve irritation
                - should be carried out for 6-12 weeks, depending on patient response
            - surgical
                - may be indicated where there is:
                    - no improvement after 6-12 weeks of conservative treatment
                    - progressive palsy or paralysis
                    - clinical evidence of long-standing lesion (e.g. muscle wasting, clawing of 4^{th} and 5^{th} digits)
        - prognosis
            - if appropriate decompression is performed in a timely manner there should be a return to normal function
            - if there is transposition of the nerve following decompression, postoperative immobilisation and rehabilitation, it may take 3-6 months
- radial nerve compression
    - background
        - least common entrapment syndrome of major nerves in the upper limb
        - may occur at any point along the anatomic course of the nerve
        - the most frequent site of compression is in the proximal forearm, in the area of the supinator muscle and involves the posterior interosseus branch
        - fractures at the junction of the proximal and middle thirds of the humerus and on the radial aspect of the wrist may also damage the nerve

        - radial nerve palsy may appear acutely at the time of injury, secondary to fracture healing or from a healing callus
    - presentation
        - characterised by palsy or paralysis of all extensors of the wrist and digits as well as the forearm supinators
        - very proximal lesions may also affect the triceps
        - numbness occurs on the dorsoradial aspect of the hand and the dorsal aspect of the radial 3 ½ digits
    - investigations
        - x-ray
            - to detect or rule out fracture, healing callus or tumours as the cause of entrapment
        - MRI
            - useful to detect tumours such as lipomas and ganglions, also aneurysms and rheumatoid synovitis
        - diagnostic procedures
            - EMGs to locate the site of injury and help the clinician monitor nerve recovery over time
                - may not be positive for 3-6 weeks following injury
    - management
        - initial medical management for 6-12 weeks to allow swelling and the palsy to subside
            - immobilisation and NSAIDs
            - functional splints help prevent contracture and improve function
        - surgical
            - surgery indicated if there is no improvement in 6-12 weeks with medical management unless caused by:
                - closed manipulation – remanipulation is indicated, proceeding to open exploration if no recovery
                - open fracture – the nerve is explored at the time of debridement
    - prognosis
        - depends on degree of injury
- uncommon nerve entrapment syndromes
    - suprascapular nerve entrapment
        - background
            - originates from the upper trunk of the brachial plexus, nerve roots C5 and C6
            - most commonly occurs at the suprascapular notch
            - the nerve becomes entrapped between the suprascapular ligament and the tiny bony suprascapular notch
            - athletes, especially those involved with shoulder movement, such as gymnasts, weightlifters and basketball players, are at increased risk
        - anatomy
            - the nerve travels with the suprascapular artery across the posterior triangle of the neck

- the artery and nerve dip under the trapezius and pass through the suprascapular notch on the superior border of the scapula
- the nerve passes through the suprascapular fossa, supplying supraspinatous muscles, before tracing along the base of the spine of the scapula to supply the infraspinatous muscle
- the nerve also supplies sensation to the posterior aspect of the shoulder joint

▪ presentation
- compression of sensory fibres causes an ill-localised, dull shoulder pain

▪ pathophysiology
- the suprascapular nerve can be injured in several places:
  - the suprascapular notch
  - between the spine of the scapula and the tendinous margin of the infraspinatous and supraspinatous muscles during extreme abduction and external rotation of the shoulder
  - compression at any point by a mass, most commonly a ganglion cyst

▪ investigations
- diagnosis is normally clinical
- MRI shows a high signal intensity in the affected nerve segment at the site of the compression because of the presence of oedema in the myelin sheath and perineurium
  - also good at detecting masses causing compression and muscle atrophy
- EMG studies may show signs of denervation of supraspinatous and infraspinatous but are invasive and technique dependent

▪ management
- conservative measures
  - educating the patient to adopt avoidance behaviours
    - seldom works in young, physically active patients
  - conditioning exercises and periodically injecting the nerve with bupivacaine and dexamethasone may accord long term relief
- surgery
  - recommended for physically active patients and those with severe and long-standing symptoms or weakness

▪ prognosis
- symptomatic improvement is expected in 95% of patients who undergo decompression, often within days of the surgery
- long term weakness and atrophy may take months to improve and some patients never regain full strength

o lateral femoral cutaneous nerve entrapment

▪ background
- sensory mononeuropathy
- entrapment causes may be intrapelvic (e.g. diverticulitis, uterine fibroids, pregnancy, abdominal tumours), extrapelvic (e.g. tight

garments or belts, trauma to the ASIS region such as seatbelt trauma, stretch from obesity or ascites) or mechanical (e.g. prolonged sitting or standing, pelvic tilt from leg length discrepancy)
        - associated with pregnancy, tight fitting clothing and obesity
        - more common in diabetics
        - rarely caused by a tumour or iliopsoas haemorrhage
    - anatomy
        - originates directly from the lumbar plexus and has root innervation from L2-3
        - the nerve runs through the pelvis along the lateral border of the psoas to the lateral part of the inguinal ligament
        - it then passes to the thigh through a tunnel formed by the lateral attachment of the inguinal ligament and the anterior superior iliac spine
            - most common site of entrapment
    - presentation
        - entrapment causes a painful mononeuropathy of the lateral femoral cutaneous nerve, a purely sensory nerve innervating the anterolateral thigh
        - the patient contains of numbness and paraesthesia of the upper lateral thigh, which may be painful
        - symptoms are usually unilateral and may be aggravated by walking or standing and helped by sitting
    - examination
        - numbness of the anterolateral thigh
        - tapping over the upper lateral inguinal ligament or extending the hip may make the paraesthesia worse
        - motor strength should be normal
    - differential diagnosis
        - femoral mononeuropathy
        - lumbosacral disc compression
    - diagnosis
        - usually clinical
        - can be confirmed by resolution of symptoms after injection of 0.5% bupivacaine one finger breadth below the ASIS
    - management
        - removing the cause where possible by losing weight or wearing loose-fitting clothing
        - steroid injection may temporarily relieve symptoms where pain is severe
        - surgical decompression may be required if symptoms persist
    - prognosis
        - pain typically resolves slowly over time, numbness may persist
- tarsal tunnel syndrome
    - background
        - compression of the posterior tibial nerve behind the medial malleolus

        - uncommon
        - normally presents with pain and paraesthesia extending distally from the ankle joint
    - anatomy and pathophysiology
        - the flexor retinaculum extends posteriorly and distally from the medial malleolus
        - the tibial passes behind the medial malleolus and beneath the flexor retinaculum bifurcating into calcaneal and plantar branches
        - several factors contribute to development of tarsal tunnel neuropathy:
            - soft tissue masses, for example lipomas, within the tendon sheath may cause compression neuropathy of the posterior tibial nerve
            - bony prominences and exostoses may contribute
            - valgus deformity of the hindfoot may contribute by increasing the tensile load on the tibial nerve
    - presentation
        - pain, paraesthesia and numbness are common
        - symptoms generally subside with rest but may not fully disappear
        - atrophy of the intrinsic foot muscles may occur but may be clinically difficult to ascertain
        - eversion and dorsiflexion may cause symptoms to increase at the endpoint of range of motion
    - differentials
        - plantar fasciitis
        - stress fractures of the hind foot
        - herniated vertebral disc
        - peripheral neuropathies (e.g. from diabetes or alcohol excess) or inflammatory arthritides
    - investigations
        - nerve conduction studies are useful but may be normal
            - sensory responses may be absent in elderly patients
        - MRI and US may be useful to look for soft tissue masses or space occupying lesion
        - x-ray may be useful for fractures, bony masses and osteophytes
    - management
        - conservative
            - local anaesthetics and soluble steroids
            - night splints with the foot in plantar flexion and varus if the patient has a valgus foot
            - surgical decompression if medical therapies fail
    - prognosis
        - approximately 75% of patients who undergo surgical decompression have pan relief
            - 25% have little or no relief
- bone tumour
    - benign

        - bone: osteoid osteoma
        - cartilage: chondroma, osteochondroma
        - fibrous tumour: fibroma
        - bone marrow: eosinophilic granuloma
        - vascular: haemangioma
        - uncertain: giant cell tumour
    - malignant
        - bone: osteosarcoma
        - cartilage: chondrosarcoma
        - fibrous tissue: fibrosarcoma
        - bone marrow: Ewing's sarcoma, myeloma
        - vascular: angiosarcoma
        - uncertain: malignant giant cell tumour
    - benign tumours
        - osteoid osteoma
            - usually <1cm in diameter and surrounded by dense osteoid
            - often occurs in young adults
            - most common sites are tibia, femur and vertebrae
            - presents with pain, often worse at night and relieved by NSAIDs
            - x-ray shows a radiolucency surrounded by dense bone
            - local excision is curative
        - osteochondroma
            - one of the most common benign bone tumours
            - sessile or pedunculated lesions arising from the cortex of a long bone adjacent to the epiphyseal plate
            - lesions can be single or multiple
            - often presents in adolescents as cartilaginous overgrowth at the epiphyseal plate
            - grows with the underlying bone
            - presents as a painless lump, or occasionally joint pain
            - excision should be considered if causing significant symptoms
            - problems include nerve compression, ankle diastasis and angular deformities
                - malignant transformation is more common with multiple osteochondromas and more proximal lesions
        - chondroma
            - lesions may be single or multiple
            - appears in tubular bones of the hands and feet
            - x-ray shows a well-defined osteopenic area in the medulla
            - lesion should be excised and bone grafted
        - giant cell tumour (osteoclastoma)
            - about 20% of primary bone tumours
            - aggressive, locally recurrent tumour with a low metastatic potential
            - found in the subarticular cancellous region of long bones; most occur in closed epiphyses around the knee joint and distal radius
            - only occurs after closure of epiphyses
            - patients are usually 20-40 years

        - more common in females
        - x-ray shows an asymmetric rareified area at the end of a long bone
            - cortex is thinned or perforated
        - treatment is by local excision and grafting often leads to recurrence
        - treatment of choice is wide excision and joint replacement
        - amputation if there is malignant or recurrent tumour
- chondroblastoma
    - rare, normally in epiphysis of long bones, e.g. hip, shoulder, knee
    - usually presents at 10-19 years with pain in the joint, muscle atrophy and tenderness
    - treatment is with curettage and bone grafting
- osteoblastoma
    - locally destructive progressive lesion commonly found in vertebrae
    - usually presents with dull aching pain
    - frequently needs biopsy to exclude malignancy
    - treatment is with curettage/bone grafting or an en bloc excision
- fibromas
    - occur in 40% of children >2 years
    - usually asymptomatic
    - treatment not usually required, except where it occupies >50% bone diameter – requires curettage/bone grafting to avoid pathological fracture
- unicameral bone cysts
    - fluid filled lesion, rare before age 3 and after skeletal maturity
    - usually present as pathological fractures and are asymptomatic before then; normally involves the proximal humerus or femur
    - treatment is to allow the fracture to heal and then aspirate and inject with methylprednisolone or bone marrow
- aneurysmal cyst
    - usually present before age 20 with pain and swelling
    - cavernous spaces filled with blood and solid lumps of tissue
    - mainly affect the femur, tibia and spine, which may lead to cord or nerve root compression
    - grow rapidly and may be confused with malignancy
    - treated with curettage/bone grafting or excision
    - there is recurrence in 20-30%, usually in the first 1-2 years after treatment and mainly in younger children
- fibrous dysplasia
    - fibrous replacement of cancellous bone – may be multiple or solitary, stable or progressive
    - usually asymptomatic
        - if involving the skull may cause swelling or exophthalmos
    - femoral involvement causes pain and a limp and may cause limb length discrepancy, bowing and pathological fractures
- osteofibrous dysplasia
    - presents in children aged 1-10 years

- usually involved the tibia with anterior swelling or enlargement of the leg; usually painless
- treatment is with excision and bone grafting, delayed until after 10 years because of higher risk of recurrence when younger

- eosinophilic granuloma
  - most common in boys aged 5-10 years, usually occurs before age 30
  - most often affects the skull with local pain and swelling, marked tenderness and warmth in the area
  - treatment is with curettage/bone grafting, low dose radiotherapy or steroid injection

- malignant bone tumours
  - osteosarcoma
    - the most common primary bone tumour in children, incidence highest in 15-19 year olds
    - male: female ratio 1.4:1
    - occurs in the metaphyses of long bones; most common sites are around the knee (75%) or proximal humerus
    - often presents as a relatively painless tumour
    - destroys bone and spreads into the surrounding tissue; rapidly metastasises to the lung
    - x-ray shows a combination of bone destruction and formation; soft tissue calcification produces a 'sunburst' appearance
    - disease free survival is 55-75% with surgery and effective chemotherapy
    - pulmonary metastases are associated with a poorer prognosis
  - Ewing's sarcoma
    - a primitive neuroectodermal tumour thought to arise from mesenchymal stem cells
    - rare, affects around 30 children per year in the UK
    - uncommon in African and Asian populations
    - median age at diagnosis is 15 years, extremely rare over 40 years
    - male:female ratio 1.5:1
    - usually presents as a mass or swelling, most commonly in the long bones of the arms or legs, pelvis or chest but also in the skull and flat bones of the trunk
    - other symptoms and signs include pain in the area of the tumour, redness in the area surrounding the tumour, malaise, anorexia, weight loss, fever (poor prognostic sign), paralysis and/or incontinence if affecting the spine and numbness or tingling as a result of nerve compression
    - investigations
      - x-ray shows bone destruction with overlying onion skin layers of periosteal bone formation
      - biopsy of the tumour site is used for diagnosis
      - CT/MRI to assess the extent of disease and local structures involved

        - bone scintigraphy to assess metastasis and response to treatment
        - staging:
            - IA – low-grade tumour found only within the hard coating of the bone
            - IB – low grade tumour extending locally into the soft tissues
            - IIA – high-grade tumour found only within the hard coating of the bone
            - IIB – high-grade tumour extending locally to the soft tissues
            - III – low- or high-grade tumour which has metastasised
    - management
        - first line treatment is chemotherapy
        - radiotherapy may be used in conjunction with surgery and/or chemotherapy, and sometimes instead of surgery where removal of the bone is not possible (e.g. spine)
        - surgery can remove part of a bone and replace it with prosthetic bone; amputation of the limb may be required if the tumour affects a long bone of the arm or leg
            - physiotherapy +/- prosthesis fitting may be required
    - prognosis
        - overall five year survival rate is 50%
        - with combined chemotherapy, radiotherapy and surgery survival is 60-70% for localised tumours and 20-40% for metastatic disease
- chondrosarcoma
    - one of the most common bone sarcomas of adulthood
    - most commonly present between 30 and 60 years
    - equal male to female ratio
    - may arise from pre-existing lesions (osteochondromas, chondromas) or can be primary
    - usually associated with dull, deep pain
    - x-ray may show invasiveness and soft tissue extension
    - two forms:
        - central: tumour in the pelvis or proximal long bones
        - peripheral: tumour in the cartilaginous cap of an osteochondroma
    - tend to metastasis late
    - wide local excision often possible
- spindle cell sarcomas
    - mixed group of malignant tumours including fibrosarcoma, malignant fibrous histiocytoma, leiomyosarcoma, undifferentiated sarcoma
    - present between 30-60 years
    - more common in men

                - usually found in the metaphysis of long bones, present with pain, have a high incidence of fracture at presentation
            - metastatic tumours
                - most common bone malignancies
                - usually multiple, may be solitary
                - most common primaries are breast, prostate, lung, kidney, thyroid
                - in children the most common metastatic lesions are from Wilm's tumour and neuroblastoma

- stress fracture
    - background
        - crack in the bone caused by repeated stresses which are individually insufficient to fracture it
        - not full thickness breaks, although they may progress to this if not managed
        - may involve the cortex only and show up poorly on x-ray
        - overuse injuries where the capacity of osteoblasts to remodel becomes exhausted
        - most common in the lower limbs
        - seen in otherwise healthy individuals
        - sports involving running or jumping carry the highest risk
        - the tibia, fibula and metatarsals are most commonly affected
    - risk factors
        - extrinsic
            - increased load
                - increased distance or frequency
                - change in training pattern – e.g. introduction of hill running, change of running surface
                - muscle fatigue - fatigued muscles lose their shock absorbency
            - increased number of load repeats
                - increased distance or frequency
            - decreased surface area over which load is applied
                - altered technique – including sedentary people who suddenly exercise
            - poor footwear
        - intrinsic
            - factors affecting bone strength (e.g. low bone density, lowered bone turnover rate – osteoporosis, osteomalacia)
            - skeletal alignment (leg length differences)
            - in women, lower muscular strength and endurance
            - hormonal abnormalities
            - previous stress fractures
            - female gender
            - body mass index <19, late menarche (≥15), previous participation in gymnastics or dance in girls
            - prior fracture and increased number of seasons of athletics in boys
            - eating disorders

- psychological factors – running with pain or inability to accept reductions or changes to training activity

- sites
  - most frequent sites:
    - tibia
    - metatarsals
    - fibula
  - sites by activity
    - track runners
      - navicular, tibia, metatarsals
    - distance runners
      - tibia and fibula
    - dancers
      - metatarsals
    - rowers
      - sometimes ribs, but rare
- presentation
  - dull bone pain that worsens with weight-bearing or repetitive use
    - usually, but not always, well-localised
  - tenderness and localised swelling may occur (apart from femoral neck, where the site is too well padded)
  - pain on activity may be worse to start with but will not necessarily prevent the activity
    - may ease during the activity and resume afterwards
    - will start to occur progressively earlier in the workout regime
    - generally decreases with rest
  - may come on for the very first time during or after exercise
  - may start suddenly after impact or come on more gradually
- diagnosis
  - high index of suspicion required
  - particular care needed with atypical stress fractures and those with a high risk of progression to complete fracture or non-union
    - neck of femur
    - middle third of tibia
    - medial malleolus
    - talus
    - tarsal navicular
    - fifth metatarsal
  - x-rays have low sensitivity and may not show the fracture until healing is well underway
    - in some cases delay in diagnosis can lead to catastrophic fracture
  - radionuclide bone scanning is highly sensitive but does not allow visualisation of fracture lines
  - bone scintigraphy is useful but involves ionising radiation so should not be used if there is an alternative (same for CT)
  - MRI is highly sensitive and specific and the method of choice where available

- management
  - rest from the aggravating activity and removal or modification of risk factors, usually for at least 4-8 weeks
    - fitness can be maintained by working out on fitness machines, water running and cycling
  - intervention at the point when the bone is under strain and remodelling is beginning to fail but before a true stress fracture occurs may be preventative
  - when there is a true stress fractures, options include rest, casting, splinting and internal fixation
  - management should begin at the point of suspicion
  - phase 1 (1-3 weeks)
    - removal of the abnormal stress
    - exercise to maintain fitness and prevent atrophy
    - safe pain-free return to usual activity
    - time for maturity of healed bone to catch up with increased bone remodelling
    - casting may be indicated when the individual cannot avoid the stressor, but should not be used routinely as it may contribute to further weakening of bone and muscle
    - crutch walking is an alternative
    - ice to decrease swelling
    - anti-inflammatories for pain
    - progression to full weight bearing as soon as it is painless, and physiotherapy in the meantime for muscle strengthening
  - phase 2 (typically 2 weeks)
    - increase exercise but stay within what they can do without pain
    - walking for at least 30 minutes three times a week, pain-free, is needed before progression to phase 3
    - if pain recurs, go back a step
  - phase 3
    - introduction of running or jogging
      - start slowly
    - increase activity no more than 15-20% per week
    - physiotherapy is continued during this phase
    - failure of treatment is most likely to occur during phase 3
    - running should be in a cycle of two weeks on, one week off for 3-6 weeks to allow bone to remodel
- high risk stress fractures
  - tibial stress fracture
    - gradual onset of localised pain on the inner aspect of the shin bone
    - fractures of the middle third of the anterior cortex are prone to non-union and internal fixation may be needed to prevent acute transverse fracture
  - talar stress fracture
    - deep ankle pain increasing with weight bearing activity

        - other symptoms may include night ache, pain during movements of the foot and ankle, pain on firmly touching the talus
        - treatment involves initial period of rest with crutches or a protective boot and exercises for flexibility, strength and balance
- calcaneal stress fracture
    - typically pain on either the inner or outer aspect of the heel bone that increases with impact activity
    - treated with an initial period of rest which may include crutches or a protective boot
- fifth metatarsal stress fracture
    - pain in the forefoot increasing with impact activity; may decrease with rest
        - may radiate to other areas of the foot
    - occasionally there is swelling or discolouration at the site
    - in more severe cases, standing or walking may aggravate symptoms and patients may experience rest pain
    - prone to non-union and progression to full fractures
    - frequently managed with non-walking short leg casts for 6 weeks, after which activity is gradually resumed
    - most patients make a full recovery; occasionally internal fixation is needed
- tarsal navicular stress fracture
    - common in runners, jumpers and basketball players
    - insidious onset of foot pain along the medial arch or along the dorsum
    - recovery is poor without treatment
    - even with rest and gradual return to activity the risk of non-union is high
    - immobilisation in a cast is recommended for at least 6 weeks, followed by a 6 week rehabilitation programme
- medial malleolar stress fracture
    - occur in distance runners
    - typically cause medial ankle pain
    - fracture line is often vertical
    - most respond to immobilisation for 6 weeks, some require internal fixation
- femoral stress fracture
    - uncommon but can have serious consequences, particularly progression to full femoral neck fracture
    - should be included in the differential when an athlete has groin pain and reduced range of movement in the hip
    - typically poorly localised pain in the front of the thigh that increases with impact and decreases with rest
    - pain may cause the patient to cease activity and may eventually be present when lying on the affected leg and at night although tenderness cannot be clinically detected
    - internal fixation is commonly considered

- most patients make a full recovery in 3-12 months, but it may take 1-2 years in more severe cases, with some having ongoing symptoms or complications

- prevention
  - gradual increase of high impact exercise (e.g. increasing distance by no more than 10%/week)
  - building muscle strength in the legs – increases shock absorption and prevents rapid fatigue
  - appropriate warm ups before exercise, including stretches
  - seeking advice if developing pain during running
  - minimise changes in shows and running surfaces by making changes gradual
  - well cushioned shoes that fit can help prevent stress fractures
    - runners should replace shoes every 300-700 miles
    - exercise technique
      - running on smooth, level surfaces such as a treadmill
      - avoid running on an injury
      - bone is weakest in the third week after the initiation of a stressful activity – consideration altered training intensity during the third week of a programme
    - general measures that prevent osteopenia and osteoporosis – avoidance of smoking, avoidance of excessive alcohol, good calcium and vitamin D intake

MuC5 spinal pain and radiculopathy

- cervical disc protrusion and lesions
  - background
  - intervertebral discs consist of the peripheral fibrocartilaginous annulus fibrosus and a the central gelatinous nucleus pulposus
  - there is no disc between C1 and C2 (atlas and axis), only ligaments and joint capsules limit excessive motion
  - disc degeneration or herniation can injure the spinal cord or nerve roots
  - cervical radiculopathy is compression or injury to a nerve root in the cervical spine
  - causes of cervical disc disorders
  - disc herniation
    - nucleus pulposus bulges or breaks through the annulus of the disc
    - repetitive injuries are most common; may occur from a single whiplash injury
    - most frequently occurs at C4/5, C5/6 and C6/7
    - posterior herniation causes symptoms by compressing the cord or a nerve root, or by stretching the posterior longitudinal ligament or posterior annulus
  - degenerative disc disease (cervical spondylosis)
    - exact cause unknown, likely to be multifactorial including genetic, environmental, traumatic, inflammatory, infectious and other factors

        - annular tears, internal disc disruption and resorption, disc space narrowing, disc fibrosis and osteophyte formation can all occur
        - degenerative disc disease may lead to disc herniation
- internal disc disruption
    - includes damage to the disc without external deformity
    - may result from whiplash or other trauma to the neck
    - degenerative disease can progress to internal disc disruption
- red flags for neck pain
- serious underlying cause more likely:
    - new symptoms before age 20 or after age 55
    - weakness involving more than one myotome or loss of sensation involving more than one dermatome
    - intractable or increasing pain
- red flags suggesting possible malignancy, infection or inflammation:
    - fever
    - unexplained weight loss
    - history of inflammatory arthritis
    - history of malignancy, drug misuse, TB, AIDS, other infection
    - immunosuppression
    - pain that is increasing, unremitting and/or disturbs sleep
    - lymphadenopathy
    - exquisite local tenderness over a vertebral body
- red flags suggesting myelopathy (compression of the spinal cord):
    - insidious progression
    - gait disturbance, clumsy or weak hands, loss of sexual/bladder/bowel function
    - Lhermitte's sign – flexing the neck causes electric shock sensations that extend down the spine and shoot into the limbs
    - upper motor neurone signs in the lower limbs – upgoing plantar reflex, hyperreflexia, clonus, spasticity
    - lower motor neurone signs in the upper limbs - atrophy, hyporeflexia
    - variable sensory changes – loss of vibration and joint position sense more evident in the hands than in the feet
- red flags suggesting severe trauma/skeletal injury:
    - history of trauma
    - previous neck surgery
    - osteoporosis or risk factors for osteoporosis
    - increasing and/or unremitting pain
- red flags suggesting vascular insufficiency:
    - dizziness and blackouts (restriction of vertebral artery) on movement, especially on extension of the neck with upward gaze
    - dizziness, drop attacks
- history
- time and mechanism of any injury
- pain:
    - distribution

        - usually unilateral but can be bilateral
        - neck pain is frequently absent in radiculopathy
    - speed of onset
        - insidious onset is usual in cervical radiculopathy, may be abrupt in acute injury
    - pain from the disc without nerve root involvement is typically vague, diffuse and distributed axially
    - activities that raise pressure in the disc, such as lifting or Valsalva manoeuvres will exacerbate symptoms
        - lying down decreases pressure in the disc and eases pain
    - driving causes vibration that aggravates disc pain
- general health including fever or unintentional weight loss
- examination
- if pain originates from the disc but there is no nerve root involvement, neurological examination will be normal
- tenderness with movement in the posteroanterior plane may suggest disc pathology
- signs of radiculopathy:
    - decreased range of neck movement
        - common with pain and spasm from any cause
    - extension and rotation increase pain
        - in Spurling's manoeuvre, the patient's neck is extended, bent laterally and held down – it elicits radicular symptoms
    - abduction sign – pain improves when the neck is flexed or on abduction of the affected arm over the top of the head
    - upper limb weakness, paraesthesia, dermatomal sensory deficit and changes to reflexes can occur
    - a herniated disc can also cause thermal changes (thermatomes) in specific distributions
- neurological features associated with cervical radiculopathy
    - C5
        - weakness of shoulder abduction and flexion and elbow flexion
        - reduced biceps reflex
        - lateral arm sensory changes
    - C6
        - weakness of elbow flexion and wrist extension
        - reduced biceps and supinator reflexes
        - sensory changes in lateral forearm, thumb and index finger
    - C7
        - weakness of elbow extension, wrist flexion, finger extension
        - reduced triceps reflex
        - sensory changes to middle finger
    - C8
        - weakness of finger flexion
        - no reflex changes

        - sensory changes to medial side lower forearm and ring and little fingers
    - T1
        - weakness of finger abduction and adduction
        - no reflex changes
        - sensory changes to medial side upper forearm and lower arm
- signs of myelopathy on examination
    - increased upper and lower limb reflexes or other upper motor neurone signs
    - upper motor neurone signs include:
        - weakness
        - spasticity
        - hyperreflexia
        - positive Babinski's sign
        - clonus
        - positive Hoffman's reflex (flicking a finger causes adjacent fingers to flex)
    - sphincter disturbances are late features of cervical and thoracic cord compression
    - cervical spine lesions can produce quadriplegia
- differentials of neck pain
- cervical spondylosis
- traumatic prolapsed intervertebral disc
- simple neck pain: acute neck strain, postural neck ache or whiplash
- headache
- referred pain (e.g. from the shoulder)
- malignancy: primary tumours, secondary deposits or myeloma
- infections: discitis, osteomyelitis, tuberculosis
- fibromyalgia
- vascular insufficiency
- psychogenic neck pain
- inflammatory disease: rheumatoid arthritis
- metabolic diseases: Paget's disease of the bone, osteoporosis
- investigations
- neck pain with radiculopathy and no red flag features does not usually require imaging or other investigations as it is likely to be self-limiting
- bloods
    - FBC for anaemia of chronic disease or evidence of infection
    - ESR/CRP
    - rheumatoid factor/HLA-B27 as indicated
- imaging
    - x-ray
        - chronic degenerative changes
        - metastatic disease
        - infection
        - spinal deformity

                - instability
            - interpretation is difficult as degenerative changes are almost universal over age 35
        - MRI
            - if no abnormality on x-ray but symptoms continuing
            - looks for intervertebral disc herniation with or without compressive or spondylotic osteophytes
        - CT myelography may be considered if there are contraindications to MRI
    - management
    - look for and treat any comorbidity
    - drug treatment
        - analgesia to relive pain and help muscle spasm
        - if pain is chronic and severe, amitriptyline or gabapentin may be added
        - diazepam for 3-7 days may be useful for severe muscle spasm
    - early mobilisation is important
    - physiotherapy including stabilisation exercises and posture training may be useful
    - heat and massage may relieve muscle spasm
    - neck supports should be used for as short a time as possible and under supervision
    - manual therapies such as manipulation have insufficient evidence
    - surgery
        - the finding of a disc lesion does not mean that surgery is indicated – in most cases conservative management is all that is needed
            - there may be spontaneous regression of a herniated disc
        - if there are significant neurological signs such as upper motor neurone limb signs or bladder disturbance, surgical decompression is indicated
        - surgery may also be indicated in intractable pain
    - prognosis
    - symptoms of cervical radiculopathy resolve in most people without surgical treatment
    - surgery has good results but is only indicated in a minority
    - the general prognosis for neck pain is not good and it is often chronic and persistent
- spinal stenosis
    - background
        - caused by narrowing of the spinal canal or neural foramina producing root ischaemia and neurogenic claudication
        - most often caused by a combination of loss of disc space, osteophytes and a hypertrophic ligamentum flavum
        - not all patients with narrowing develop symptoms
            - it may progress to cause compression of the spinal nerves and/or cord compression
        - most often affects the lumbar and/or cervical spine

        - symptomatic stenosis of the thoracic spine is very uncommon
- cervical spine stenosis
    - occurs in 9% of people over 70
    - the upper cervical segments are only rarely affected
    - the most characteristic symptoms are:
        - neck pain with restricted range of movement of the neck
        - instability of gait
        - loss of fine motor control of the upper limbs
        - weakness and sensory disturbance in the upper and lower limbs
        - urinary urge incontinence
    - examination may reveal motor and sensory disturbance in the upper limbs and upper motor neurone signs in the lower limbs
    - conservative treatments include
        - physiotherapy
        - epidural injections
        - NSAIDs
        - other medications for pain relief as required
    - surgical options include:
        - anterior discectomy and fusion
        - anterior corpectomy and fusion
        - arthroplasty
        - posterior laminectomy with or without fusion
        - laminoplasty
- lumbar spinal stenosis
    - background
        - symptomatic lumbar stenosis is thought to occur in around 10% of the population
    - risk factors
        - degenerative arthritis of the spine
        - congenital narrowing of the spinal canal
        - hyperparathyroidism
        - Paget's disease of the bone
        - ankylosing spondylitis
        - Cushing's syndrome
        - acromegaly
    - presentation
        - clinical syndrome of pain in the buttocks or lower extremities with or without back pain
            - often exacerbated by standing, walking or lumbar extension
            - relieved by forward flexion, sitting or lying flat
        - gradual onset of unilateral or bilateral leg pain (with or without back pain), numbness and weakness developing after walking a predictable distance
            - patients may have less difficulty walking uphill than downhill
        - about half of patients present with back pain, usually bilateral and diffuse over the buttocks

                - neurogenic intermittent claudication: leg fatigue and/or weakness and leg numbness and/or paraesthesiae
                - pain:
                    - bilateral leg pain with burning or cramping; involves the buttocks and thighs and spreads to the feet
                    - the neural canal and foramen are narrowed with the spine in backward extension and opened in forward flexion – neural compression is usually intermittent and provoked by lying prone or arching the lumbar spine and when upright, particularly walking
                    - cycling does not usually cause significant problems
                    - pain is usually relieved by sitting, leaning forward, putting the foot on a raised cushion or stool or lying supine
            - a complete motor and sensory neurological examination is required and is often normal
            - lower limb vascular examination is required to rule out vascular claudication
            - differential
                - cauda equina syndrome
                - peripheral arterial disease
                - spinal tumours
                - large central disc herniation
                - spondylolisthesis
                - lumbar spine trauma or vertebral fracture
                - epidural abscess
                - inflammatory arachnoiditis
            - investigations
                - x-ray
                    - initial assessment for possible alternative diagnosis
                - lumbar spine MRI or CT (MRI preferred)
    - management
        - conservative
            - weight reduction if overweight
            - physiotherapy with forward flexion exercises
            - NSAIDs and other medication for pain relief as appropriate
            - epidural injections of local anaesthetic or local anaesthetic with steroids can offer long and short term relief
            - medications for neuropathic pain may be needed
        - surgery
            - occasionally indicated for patients not responding to conservative measures
    - prognosis
        - prognosis with conservative treatment is generally good for most patients

MuC6 risks of rheumatological disease modifying drugs

- background
    - broadly speaking, they either affect the immune response or suppress the disease process

    - most require regular blood test monitoring
- types of DMARD
    - conventional DMARDs or non-biological DMARDs
        - e.g. methotrexate, leflunomide, sulfasalazine
        - continue to be first line treatments
        - hydroxychloroquine sulphate, a weak DMARD, may be used in patients with milder symptoms
    - biologic and targeted synthetic DMARDs
        - used in severe conditions where intensive treatment with conventional DMARDs has not suppressed the disease
        - may be given in combination with a conventional DMARD
        - biologics include:
            - anti-tumour necrosis factor agents
                - e.g. adalimumab, etanercept, infliximab
            - other biologic agents
                - e.g. abatacept, tocilizumab
            - targeted synthetic DMARDs – Janus kinase (Jak) inhibitors
                - e.g. tofacitinib, baricitinib
- general principles for use
    - initiated by specialists
    - all DMARDs except hydroxychloroquine require regular blood tests
    - caution for interactions with other medications
    - people on DMARDs are more prone to infections and complications of infections
        - there is more risk of toxicity during intercurrent illness, particularly if there is renal impairment
    - live vaccines (e.g. MMR, oral typhoid, yellow fever) are contraindicated
        - routine vaccination with non-live vaccines against common infections should be given
    - specialists should screen for TB, hepatitis B, hepatitis C and HIV where risk factors are present prior to starting DMARDs
    - people on DMARDs should be aware of potential adverse effects and know who to contact should they develop
- concerning blood results
    - results which should prompt immediate discussion with the specialist team whilst withholding the DMARD include:
        - WCC <3.5
        - neutrophils <1.6
        - eosinophils >0.5
        - MCV >105
        - platelets <140
        - creatinine increase of >30% over 1 year and/or calculated GFR <60
        - ALT and/or AST >100
        - unexplained reduction in albumin <30
        - 2+ or more urinary protein in the absence of urinary infection
- complications and reasons to discontinue drugs
    - potential side effects include:
        - myelosuppression (all DMARDs)

        - renal toxicity
        - liver toxicity
        - pulmonary problems
        - skin rashes
        - gastrointestinal disturbance
        - there may be increased risk of skin cancers (conflicting evidence)
        - anti TNF therapy may potentially worsen or cause demyelination
    - patients should be warned about symptoms such as:
        - sore throat
        - fever and other signs of infection
        - unexpected bleeding or bruising
        - purpura and rashes
        - mouth ulcers
        - cough or breathlessness
        - peripheral neuropathy
        - nausea or vomiting
        - abdominal pain
        - dark urine
    - other concerning symptoms – stop DMARD and refer urgently to rheumatology:
        - symptoms of possible TB (cough, haemoptysis, weight loss)
        - signs or symptoms of heart failure, or worsening heart failure
        - symptoms of interstitial lung disease (e.g. shortness of breath or dry cough)
        - skin rashes
        - severe abdominal pain or change in bowel habits accompanied by fever

MuC7 spinal infections

- epidural abscess
    - background
        - spinal infection caused by a collection of pus or inflammatory granulation tissue between the dura mater and surrounding adipose tissue
        - early diagnosis is critical and made with contrast MRI
        - treatment is usually prompt surgical decompression and long term IV antibiotics
        - usually seen in adults >60, usually in the thoracolumbar spine
    - risk factors
        - IV drug abuse
        - immunodeficiency
        - malignancy
        - HIV
        - immunosuppressive medication
        - recent spinal procedure
    - pathophysiology
        - origin
            - haematogenous spread (50%)
            - spread from discitis (33%)
        - pathogens
            - Staph aureus most common (50-65%)

- Gram negative infections such as E coli
- pseudomonas in patients with IV drug abuse

- presentation
  - symptoms
    - systemic illness more profound than patients with vertebral osteomyelitis
      - fever in 50%
    - pain
      - often severe and insidious in onset, occurs in 87%
  - physical examination
    - neurological deficits present in 33%
      - may present as a radiculopathy or myelopathy
      - 4-22% incidence of permanent paralysis
        - can be caused by direct compression or infarction of spinal cord blood flow
- investigations
  - bloods
    - WCC (raised in 42%)
    - ESR (raised in 90%)
    - CRP (raised in 90%)
  - imaging
    - x-rays
      - usually normal
    - CT
      - poor sensitivity
    - CT myelogram
      - 90% sensitivity but invasive
    - MRI with gadolinium
      - imaging modality of choice
      - shows extent of abscess, presence of vertebral osteomyelitis, evaluation of neurological compression
      - allows differentiation of pus from CSF
      - entire spine should be imaged to rule out skip abscesses
- management
  - non-operative
    - bracing and IV antibiotics
      - indications
        - small abscess with minimal compression, no neurological deficit and patient capable of close clinical follow up
        - those not suitable for surgery due to comorbidities
      - medical treatment failure associated with:
        - neurological deficit
        - diabetes
        - CRP >115
        - WCC >12
        - positive blood cultures

                - age >65 years
                - MRSA
        - operative
            - surgical decompression +/- spinal stabilisation
                - indications
                    - neurological deficit present
                    - evidence of spinal cord compression on imaging studies
                    - persistent infection despite antibiotic therapy
                    - progressive deformity or gross spinal instability
                - postoperative antibiotics
                    - 2-4 weeks if no bony involvement of infection
                    - 6 weeks if bony involvement
    - prognosis
        - preoperative degree of neurological deficit is most important indicator of clinical outcome
        - mortality is around 5%
        - early diagnosis is the most essential factor in preventing devastating outcomes
- discitis
    - background
        - infection in the intervertebral disc space
        - infection of nucleus pulposus due to infection of vertebral body end plate cartilage
    - risk factors
        - paediatric patients (<8 years)
        - post-operative spinal surgery
        - immunocompromised
    - clinical features
        - >90% present with unremitting neck or back pain which awakens them at night
            - may have radicular symptoms
        - fever (60-70%)
        - neurological deficit (10-50%)
        - raised ESR (>90%)
        - leucocytosis (<50%)
    - investigation
        - bloods
            - raised ESR
        - imaging
            - x-rays rarely useful
            - MRI is gold standard
    - management
        - IV antibiotics
- vertebral osteomyelitis
    - background
        - usually have prolonged symptoms (>3 months of pain)

        - fever is a feature but they are often afebrile
        - vertebral body tenderness
        - paravertebral muscle spasm unresponsive to conservative therapy
        - paravertebral or epidural abscess may develop
    - investigation
        - ESR – almost always elevated
        - blood cultures
        - imaging
            - may take 2-8 weeks to see changes
            - bony destruction, irregularity of vertebral end plates, disc space narrowing
    - management
        - antibiotics
            - may require biopsy before starting antibiotics – advice from spinal surgeon needed
            - usually IV antibiotics for 6 weeks then PO for 4-8 weeks
            - e.g. Tazocin and vancomycin

MuC8 torticollis

- background
    - also known as 'wry neck'
    - is a twisted neck
    - acute torticollis is thought to be due to minor local musculoskeletal irritation causing pain and spasm in the neck muscles
    - acute acquired torticollis is common
        - causes include:
            - sleeping in an awkward position
                - symptoms usually resolve spontaneously in 1-2 days and last no more than 1-2 weeks
            - upper respiratory and soft tissue infections
                - can cause an inflammatory torticollis secondary to muscle contracture or adenitis
            - any abnormality or trauma of the cervical spine
            - may be due to bad posture, e.g. poor positioning at a computer screen, inappropriate seating, sleeping without adequate neck support, carrying heavy unbalanced loads
- presentation
    - sudden onset (often on waking) of severe unilateral pain with deviation of the neck to that side
        - they may have some premonitory twinges
        - occasionally the pain may be in the middle of the neck
        - pain may be referred to the head or shoulder region
    - the neck feels stuck in one position and any attempted movement to free it results in sharp spasms of pain
    - no history of trauma or strain

    - a history of localised exposure to cold, prolonged or unusual positioning of the neck or unusual posture (e.g. holding the neck in an unusual position whilst working, reading or sleeping)
- signs
    - tenderness is usually diffuse on the involved side with palpable spasm
    - look for trigger points (tender points of muscle spasm)
    - check for restricted or painful movement
    - visual acuity testing and full eye examination should be considered, especially in children presenting with torticollis
- management
    - advise that acute torticollis usually resolves in 24-48 hours
        - occasionally symptoms may take up to a week to resolve and recurrence is common
    - analgesia, depending on the severity of pain
        - usually paracetamol or ibuprofen
        - codeine as second line
    - gentle exercise, intermittent heat or a cold pack
    - sleeping on a low firm pillow
    - maintaining good posture
    - causes should be identified and treated accordingly
    - advised against use of a soft cervical collar
    - advise against driving as it is not possible to rotate the head to view traffic
- other causes of neck pain and muscle spasm
    - acute disc prolapse
        - most common cause of severe secondary torticollis
    - tonsillitis, retropharyngeal abscess
    - cervical lymphadenopathy due to infection or cancer
    - vertebral infection (e.g. osteomyelitis)
    - cervical spine injury (e.g. traumatic fracture or dislocation)
    - eye disorders
    - dystonia due to any underlying condition (e.g. stroke, encephalitis, cervical dystonia)
    - drug dystonic reactions
    - pseudodystonias (e.g. vestibular disorder, tumour of the posterior fossa)
    - somatisation

MuC9 limping child

- important points about limping children
    - consider the age
        - key in forming a differential
    - most are ok
        - the majority have a benign and self-limiting condition
    - investigate fever
        - any child with a fever and limp warrants further investigation
    - consider referred pain
        - consider non-musculoskeletal causes such as referred testicular or abdominal pain
    - ask about systemic symptoms

        - suggests serious underlying disease
- common causes of limp in children
    - any age
        - trauma
            - fracture
            - haemarthrosis
            - soft tissue
        - infection
            - septic arthritis
            - osteomyelitis
            - discitis
        - secondary to various viral illnesses
        - tumour
        - sickle cell disease
        - serum sickness
    - toddler (1-3 years)
        - transient synovitis
        - toddler's fracture
        - child abuse
        - developmental dysplasia of the hip
        - juvenile arthritis (pauciarticular)
        - neuromuscular disease
        - haemophilia
        - Henoch-Schönlein purpura
    - child (4-10 years)
        - transient synovitis
        - juvenile arthritis (pauciarticular)
        - Perthes' disease
        - rheumatic fever
        - haemophilia
        - Henoch-Schönlein purpura
    - adolescent (11-16 years)
        - slipped upper femoral epiphysis
        - overuse syndromes
        - osteochondritis dissecans
- transient synovitis
    - background
        - also known as irritable hip
        - more common in boys
        - often preceded by viral infection
        - benign condition with a small amount of fluid in the hip
        - no reliable way of differentiating from septic arthritis
        - clinical suspicion and blood tests are important (Kocher's criteria)
    - presentation
        - typically pain of acute onset followed by spontaneous recovery with no systemic upset
        - pain is not severe but may prevent weight-bearing

        - usually no pain at rest, and passive movement is only painful at the extremes
        - child is usually systemically well
        - ESR is normal or mildly raised
    - management
        - rest
        - oral analgesics
        - observation
        - mobilisation once pain settles
    - prognosis
        - self resolves
        - recurs in up to 15% of children
- septic arthritis
    - background
        - children appear ill
        - usually in the hip
        - the hip is very vascular so haematogenous spread can occur easily
        - most are under 4
        - most common causative agent is Staph aureus
    - presentation
        - fever
        - pain
        - irritability
        - inability to weight bear
        - hip often held flexed and abducted
    - investigation
        - clinical prediction algorithm uses
            - history of fever
            - non weight-bearing
            - ESR of at least 40
            - WCC >12
        - predicted probability >90% for 3 or 4 predictors
    - management
        - surgical emergency
        - requires early surgical drainage and IV antibiotics to prevent bony destruction and preserve hip function
- toddler's fracture
    - background
        - also known as Childhood Accidental Spiral Tibial (CAST) fracture
        - non-displaced or minimally displaced spiral fracture of the tibia
        - typically in ambulating toddlers (9 months to 3 years) due to low energy trauma with rotational component
        - not generally associated with non-accidental trauma
    - clinical features
        - otherwise healthy child, who was ambulating, falls and then is non-ambulatory or has painful ambulation
        - usually pain with palpation and rotation of the distal tibia

        - swelling may be minimal or absent
    - investigation
        - x-ray
            - may present only as a faint oblique line on AP view
            - may require oblique view in addition
    - management
        - if clinical suspicion but no fracture seen on x-ray:
            - casting/soft wrap
            - review with repeat imaging in 1 week
        - if fracture seen:
            - long leg splint or cast and orthopaedic referral
            - usually heals in 3-4 weeks without intervention
- developmental dysplasia of the hip
    - background
        - formerly known as congenital dislocation of the hip
        - affects 1-3% of newborns
        - responsible for 29% of primary hip replacements in people under 60
        - left hip is dislocated more commonly than the right
        - 20% of cases are bilateral
        - less common in cultures that carry their babies in a straddle position with hips widely abducted
    - risk factors
        - having a sibling with dysplasia increases risk by 5%
        - 80% of cases are in girls
        - about 60% are first born
        - vaginal delivery of breech presentations has a 17 fold increased risk
        - restriction of movement with oligohydramnios increases risk
        - risk increased in multiple pregnancy and prematurity
        - more common with neuromuscular disorders
    - screening
        - part of the physical examination of the newborn (appearance, limb length discrepancy, Barlow and Ortolani tests) and 6-8 week examination
        - US screening should be performed in specific circumstances:
            - first degree family history of hip problems in early life unless DDH definitely excluded
            - breech presentation
                - at or after 36 weeks irrespective of presentation at delivery or mode of delivery
                - at delivery if earlier than 36 weeks
                - in multiple birth if any of the babies falls into one of these categories – all should be screened
    - management
        - early diagnosis and treatment of those most severely affected is important for good outcome
        - most unstable hips stabilise spontaneously at 2-6 weeks of age and any hip dislocatable or pathologically unstable after this time requires prompt treatment

        - bracing is first line treatment in children <6 months
            - Pavlik harness
                - left in place at all times to maintain hip reduction
                - main risks are avascular necrosis and temporary femoral nerve palsy
        - surgery is an option for older children or for those in whom non-operative management has failed
    - follow up
        - required at least until the hip is clinically stable and imaging shows a stable, centred normal hip
- juvenile arthritis
    - background
        - joint inflammation presenting in children under 16 and persisting for at least 6 weeks, with other causes excluded
        - seven subsets:
            - oligoarticular JIA (50%)
            - polyarticular JIA – RF negative (25%)
            - polyarticular JIA – FR positive (5%)
            - systemic onset JIA (5-10%)
            - juvenile psoriatic arthritis (2-15%)
            - enthesitis-related arthritis (2-10%)
            - undifferentiated arthritis (1-10%)
    - presentation
        - oligoarticular JIA
            - affects 1-4 joints in the first six months
            - 70% are ANA positive
            - usually presents under 6
            - more common in females
            - typically presents with one or two swollen joints causing stiffness and reduced movement but often not much pain
            - child usually feels well
            - knee and ankle are commonly affected
    - polyarticular JIA (RF negative)
        - affects 5 or more joints in first 6 months
        - two peaks: toddler/preschool and pre-adolescent
        - more common in females
        - can be asymmetrical (with higher risk of uveitis)
        - can be symmetrical
        - can present with stiffness but minimal swelling
        - often a destructive arthritis
    - polyarticular JIA (RF-positive)
        - affects 5 or more joints in first 6 months with positive RF on two occasions
        - symmetrical involvement of small joints, particularly hands and wrists, with swelling and stiffness
        - rheumatoid nodules may develop
        - systemic symptoms: fever, enlarged liver and spleen, lymphadenopathy, serositis, pericardial effusion

- systemic JIA
    - arthritis with at least 2 weeks of daily fever (>39 degrees)
    - one or more of:
        - rash (quickly fading, non-fixed, erythematous
        - lymph node enlargement
        - hepatomegaly and/or splenomegaly
        - serositis (pericarditis, pleuritis, peritonitis)
    - affects males and females equally
    - age of onset usually before 5 years
- juvenile psoriatic arthritis
    - arthritis and psoriasis or arthritis plus at least two of:
        - dactylitis
        - nail pitting or onycholysis
        - psoriasis in first degree relative
    - affects female twice as commonly as males
    - mean age of onset 6 years
    - asymmetrical, affecting small and large joints
    - in over 50% arthritis occurs before psoriasis
- enthesitis-related JIA
    - arthritis or enthesitis (inflammation at the site of tendon/ligament insertion) plus two of:
        - sacroiliac or lumbosacral pain
        - HLA B27 positive
        - family history of HLA B27 related disease
        - acute anterior uveitis
        - onset in male over age of 6
    - affects males nine times more commonly than females
    - age of onset usually over 10
    - often affects a lower limb, heel pain may be a symptom
    - soft tissue swelling of the knee or foot is a common examination finding
    - assess for signs and symptoms of associated psoriasis, uveitis, inflammatory bowel disease
- investigations
    - bloods, including inflammatory markers and immunology
    - imaging
- management
    - physiotherapy and occupational therapy
    - encourage to participate in as much physical activity as possible
    - drugs: NSAIDs for symptomatic relief, steroids, methotrexate, sulfasalazine or leflunomide; etanercept or tocilizumab if methotrexate ineffective
- complications
    - joint deformities
    - uveitis – can lead to blindness and is insidious and asymptomatic; all children require regular screening
    - macrophage activation syndrome
    - secondary amyloidosis
    - osteoporosis

        - growth restriction
        - complications from medications
        - restriction of sports and other leisure activities
        - psychosocial, behavioural and educational difficulties
- Perthes's disease
    - background
        - self-limiting hip disorder caused by varying degrees of ischaemia and subsequent necrosis of the femoral head
            - the essential lesion is avascular necrosis of the nucleus of the proximal femoral epiphysis
            - abnormal growth of the epiphysis results
            - there is eventual remodelling of regenerated bone
    - epidemiology
        - usually seen in 3 to 10 year olds
        - incidence of 1 in 10,000 children under 15
        - bilateral in 15%
        - more common in males
        - rare in non-Caucasians
    - presentation
        - onset is usually over weeks with no history of trauma
        - child typically presents with limitation of hip rotation and a subacute limp
            - sometimes with referred pain to groin, thigh or knee
        - typically unilateral
        - child is systemically well, with no evidence of joint inflammation
        - on examination all movements at the hip are limited
        - there is an antalgic gait and a Trendelenburg gait in the late phase
    - investigations
        - may include
            - FBC, ESR
            - x-rays (including frog leg views)
                - may show widening of the joint space
                - later there is decrease in the size of the nuclear femoral head
                - later still there may be collapse and deformity of the femoral head with new bone formation
            - technetium bone scans or MRI may be required
    - management
        - non-operative
            - restriction of activities and weight bearing until ossification is complete
            - physiotherapy
            - NSAIDs
            - good outcomes in 60% of patients with non-operative techniques
        - operative treatments
            - femoral or pelvic osteotomy
            - valgus or shelf osteotomies
            - hip arthroscopy

        - hip arthrodiastesis
    - prognosis
        - normally good but pain, osteoarthritis and ongoing hip dysfunction are common
        - at least 50% do well with no treatment
        - there is a risk of developing later osteoarthritis with need for hip replacement in the sixth decade
- slipped upper femoral epiphysis (SUFE)

https://radiopaedia.org/cases/7228/studies/8174?lang=us&referrer=%2Farticles%2Fslipped-upper-femoral-epiphysis%3Flang%3Dus%23image_list_item_32143

    - background
        - often atraumatic or associated with a minor injury
        - four separate clinical groups:
            - pre-slip: wide epiphyseal line without slippage
            - acute form (10-15%): slippage occurs suddenly, normally spontaneously
            - acute-on-chronic: slippage occurs acutely where there is already existing chronic slip
            - chronic (85%): steadily progressive slippage
        - also categorised as stable or unstable
            - stable (90%) – patient is able to walk and osteonecrosis is rare
            - unstable (10%) - patient is unable to walk, even with crutches and there is a 50% incidence of osteonecrosis
    - epidemiology
        - incidence 10 in 100,000 children/year
        - most commonly occurs in boys at 13 years and girls at 11.5 years
        - left hip more commonly affected than right
        - 1.5 times more common in boys
    - risk factors
        - mechanical
            - local trauma
            - obesity
                - more than 80% of children diagnosed with SUFE are obese
        - inflammatory

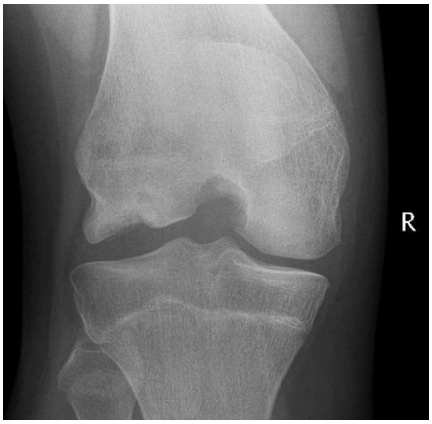
        - neglected septic arthritis
    - endocrine
        - hypothyroidism
        - hypopituitarism
        - growth hormone deficiency
        - pseudohypoparathyroidism
        - vitamin D deficiency
    - previous radiation of the pelvis, chemotherapy, renal osteodystrophy-induced bone dysplasia
    - contralateral SUFE
        - risk reduced with weight loss to lower than 95th centile
- presentation
    - discomfort in the hip, groin, medial thigh or knee (referred) during walking
    - limp
    - pain accentuated by running, jumping or pivoting activities
    - pre-slip: slight discomfort or found on x-ray
    - acute:
        - presents within 3 weeks of onset of symptoms
        - severe pain such that the child is unable to walk or stand
        - alterations in gait, including a limp on the affected side, external rotation of the leg and trunk shift
        - hip motion limited due to pain, especially internal rotation and abduction
        - obligate external hip rotation – when the child is supine and the hip is passively flexed and then falls back into external rotation and abduction
    - acute on chronic:
        - pain, limp and altered gait for several months, suddenly becoming very painful
    - chronic:
        - presents more than 3 weeks after the onset of symptoms
        - mild symptoms, with the child able to walk with altered gait
        - knee pain is the only reported symptom in a significant number of cases
        - external rotation of the leg during walking
        - reduced internal rotation with additional external rotation
        - when flexed up, the hip tends to move in an externally rotated position
        - mild to moderate shortening of the affected leg
        - atrophy of the thigh muscle may be noted
- investigations
    - AP and frog leg x-rays
        - show widening of the epiphyseal line or displacement of the femoral head
        - earliest findings include globular swelling of the joint capsule, irregular widening of the epiphyseal line and decalcification of the epiphyseal border of the metaphysis

                - the epiphysis normally extends slightly cephalad to the upper border of the femoral neck
                - small amounts of slippage can be detected by the epiphyseal edge becoming flush with the superior border of the neck
            - CT may be indicated if complex surgery is planned
        - management
            - avoid moving or rotating the leg
            - the patient should not be allowed to walk
            - analgesia and immediate orthopaedic referral if the diagnosis is suspected
            - surgery is the standard treatment but management remains controversial
                - may be minimally invasive or open
        - complications
            - chondrolysis (degeneration of the articular cartilage)
            - avascular necrosis of the epiphysis
            - recurrence or progression
            - long term effects of altered femoral head anatomy leading to osteoarthritis
        - prognosis
            - depends on the initial degree of epiphyseal slippage and on prompt recognition
            - end result is good to excellent in 91-95% of cases where slip is mild or moderate
                - with increasing displacement, complications increase

MuC10 osteochondritis

- background
    - osteochondritis dissecans is a pathological process affecting the subchondral bone (most often in the knee joint)
        - involves the separation of articular cartilage and subchondral bone fragment from a joint surface
    - it can lead to secondary effects on joint cartilage and degenerative changes if left untreated
    - more common in males
    - misnamed in the 19th century as it was thought there was an underlying inflammatory pathology
    - the cause is unknown
    - two types:
        - juvenile
            - occurring with an open epiphyseal plate
        - adult
            - after the physis has closed
- distribution
    - 75% knee joint
        - 85% of these are on the medial femoral condyle
    - elbow and ankle next most commonly affected
        - in the elbow it affects the capitellum of the humerus
        - in the ankle, it affects the talar dome
    - very rarely affects articulations of the shoulder, hand, wrist or hip

    - can affect more than one site, may be bilateral in 20-30% of cases
- risk factors
    - trauma (about half of cases)
    - male sex (incidence increasing in women and girls)
    - overuse due to sporting activity
    - familial pattern in about 10%
    - ligamentous weakness
    - genu valgum/varum
    - meniscal lesions in the knee
- presentation
    - symptoms
        - usually presents in teens or early 20s
        - can affect younger children who are very active at sports
        - may only become symptomatic in later life
            - around 5% of middle aged patients with osteoarthritis of the knee are thought to have had osteochondritis earlier in life
        - the usual features is vague, aching joint pain and swelling worsened by activity
        - locking, catching and giving way may be present, particularly with intra-articular loose bodies
        - when the lateral femoral condyle is affected, patients commonly feel a painful clunk when flexing or extending the knee
    - signs
        - full range of movement without signs of ligamentous instability in most cases
        - joint effusion is often present, particularly if there has been trauma
        - with medial femoral involvement, external tibial rotation when walking is typical
        - with the knee fully flexed, it should be possible to palpate the area directly on the articular cartilage of the medial femoral condyle, which is usually tender
        - Wilson's sign (limited evidence) for demonstrating the presence of a medial femoral condyle lesion
            - with the knee flexed to 90 degrees and the tibia internally rotated, gradual extension of the joint leads to pain at about 30 degrees
            - external rotation of the tibia at this point relieves the pain
- diagnosis
    - early diagnosis is vital
    - clinical findings can be subtle – low threshold required for ordering x-rays or seeking an orthopaedic opinion
    - juvenile lesions are typically stable with an intact articular surface and have the potential to heal with conservative management if detected early
- investigations
    - x-ray



https://radiopaedia.org/cases/3194/studies/6455?lang=us&referrer=%2Farticles%2Fosteochondritis-dissecans%3Flang%3Dus%23image_list_item_614#findings

        - subchondral crescent sign or loose bodies
        - knee requires AP, lateral and tunnel (with knee in flexion) views
    - ultrasound
        - may be useful and is cost-effective, allows dynamic imaging with joint movement
    - CT
        - demonstrates size and site of lesion
    - MRI
        - best for evaluation of overlying cartilage
        - used to stage and assess stability of the lesion
        - useful for prognosis
    - scintigraphy
        - may show increased uptake in the fragments
        - osteoblastic activity is useful to guide treatment – relates to a greater chance of healing with conservative management
- staging (by MRI appearance)
    - I
        - thickening of articular cartilage and low signal changes
        - stable lesion
    - II
        - articular cartilage interrupted, low signal rim behind fragment showing that there is fibrous attachment
        - stable lesion
    - III
        - articular cartilage interrupted, high signal changes behind fragment and underlying subchondral bone
        - unstable lesion
    - IV
        - loose body
        - unstable lesion
- management
    - no good evidence from randomised controlled trials

    - approaches take into consideration the maturation of the growth plate, situation of the subchondral bone, stability of the lesion, dimensions of the fragment and integrity of the cartilage
    - conservative treatments
        - analgesia/anti-inflammatories
        - load reduction (crutches)
        - use of an immobiliser
        - gentle physiotherapy
        - plaster casts
            - may predispose towards chondral degeneration and joint stiffness
    - total restriction of physical activity may lead to resolution of the process in younger patients
    - surgery
        - indicated where conservative treatment fails, for loose bodies and in cases of unstable or dislocated lesions, especially for adults
- complications
    - pain
    - functional impairment
    - knee joint effusions
    - loose body formation
    - osteoarthritis
- prognosis
    - depends on age of patient, affected joint and stage of lesion at presentation
    - younger patients with small, stable medial femoral condyle lesions have the best prognosis
    - unstable lesions can heal after stabilisation, long term prognosis is not clear
    - chronic loose fragments can be difficult to fix and heal poorly
    - excision of large lesions from weight bearing zones also tends to give poor results

NEONATAL EMERGENCIES

NeoC1 delivery and resuscitation of the newborn

- stages of labour
    - first (dilatation)
        - latent
            - passage of cervical mucus plug
            - slow dilation to 6cm
        - active
            - rapid cervical dilation from 6cm to 10cm
        - lasts around 8 hours in primiparous and 5 hours in multiparous
    - second (birth)
        - cervix fully dilated
        - urge to push
        - expulsion of infant
        - around 20-50 minutes
    - third (placental delivery)

        - around 20 minutes
        - >18 minutes increases risk of postpartum haemorrhage (>30 minutes greatly increases risk)
    - fourth (post-placental delivery)
        - first hour after placental delivery
        - period with highest risk for postpartum haemorrhage
- six cardinal movements of foetal descent
- engagement
- flexion
- descent
- internal rotation
- extension
- external rotation
- clinical features of labour
    - abdominal pain
    - rupture of membranes
        - pooling of fluid in the vaginal vault
        - ferning pattern when fluid allowed to dry on microscope slide
        - pH testing with nitrazine paper turning blue
    - crowning
- evaluation
    - cervical dilatation
        - diameter of the internal cervical os increases as labour progresses
        - 0cm (closed/fingertip) to 10cm (complete/fully dilated)
            - measure with index and middle fingers of examining hand
            - use sterile gloves, sterile lubrication and sterile speculum
    - effacement
        - assessment of the cervical thinning
        - percentage of normal 3-4cm long cervix
            - 4cm cervix = 0%
            - 0cm (thin) cervix = 100%
        - fully effaced cervix feels paper thin
    - station (-5 to +5)
        - distance of the presenting body relative to the maternal ischial spines
            - -3 = beginning of second stage of labour
            - 0 = in line with the plane of the maternal ischial spines
            - +3 = impending delivery
            - +4 to +5 = crowning
    - true vs false labour
        - false labour is defined as uterine contractions that do not produce cervical changes
        - Braxton-Hicks contractions
            - brief contractions, irregular in both duration and intensity
        - true labour is characterised by regular contractions that lead to cervical changes, gradually increasing in intensity and duration
- management
    - preparation

        - position patient in dorsal lithotomy position
        - put on personal protective equipment
        - prepare suction, airway equipment and warmer for infant
        - call for obstetrics, NICU and paediatrics
        - call for additional staff members
        - divide team into maternal team and infant team
    - second stage
        - perineal inspection
            - infant's head bulges the perineum
            - if prolapsed cord is present, elevate the presenting foetal part, place patient in Trendelenburg position and call obstetrics immediately
            - gentle digital stretching with a lubricated finger may prevent tears and lacerations
            - support the perineum with a sterile towel and place the other hand over the occiput to promote foetal head extension
        - slowly deliver the head
            - check for nuchal cord
                - if present, slip finger between infant's neck and cord and attempt to reduce cord by pulling over infant's head
                - if unable to reduce cord, clamp or cut cord if infant's face can be cleared from perineum with immediate suction
        - deliver anterior shoulder
            - position hands on either side of the head and exert a gentle downward force
        - deliver posterior shoulder
            - maintain position of hands and apply a small amount of upward traction
        - delivery of the body
            - controlled expulsion helps to prevent perineal lacerations
    - after delivery of infant
        - hold infant securely
        - position in a manner that facilitates the flow of blood from the placenta to the infant
        - stimulate and dry the infant
        - clamp then cut the umbilical cord 3cm distal to insertion at umbilicus with sterile scissors
            - ideally delayed 30-60 seconds after delivery
        - if uncomplicated delivery with clear airway and good respiratory effort, mother may hold child immediately (skin to skin)
        - if mother or infant is unstable, pass infant to receiving team
            - place infant in a warm incubator
            - check APGAR scores at 1, 5 and 10 minutes after delivery
    - third stage
        - maintain manual suprapubic pressure
        - provide gentle cord traction and allow spontaneous placental separation
        - placenta usually delivers within 10-30 minutes
            - avoid excessive cord traction to prevent uterine inversion

                - signs of placental separation:
                    - abrupt lengthening of cord
                    - sudden gush of blood
                    - cephalad migration of uterus
                - inspect for missing placental segments and normal cord insertion and vessels
                    - normal cord should have 3 vessels
                    - if placenta is not intact, there may be retained products of conception requiring manual or surgical removal
            - ergometrine with oxytocin (Syntometrine) 1ml IM
                - helps to prevent postpartum haemorrhage
    - fourth stage
        - first hour after placental delivery
        - palpate abdomen and check for achievement of uterine firmness and contraction
        - period of time with highest risk for postpartum haemorrhage (>500ml of blood)
- complications
    - amniotic fluid embolus
    - chorioamnionitis
    - acute fatty liver of pregnancy
    - eclampsia
    - HELLP syndrome
    - mastitis
    - peripartum cardiomyopathy
    - postpartum endometritis
    - postpartum headache
    - postpartum haemorrhage
    - pre-eclampsia
    - resuscitative hysterotomy
    - retained products of conception
    - uterine rupture
- newborn life support (Resus Council)
    - team briefing and equipment check
    - birth
        - delay cord clamping if possible
            - benefits include:
                - avoidance of bradycardia and abrupt cardiovascular changes
                - improved early haemoglobin
                - improved iron stores in infancy
                - in preterm infants:
                    - improved survival
                    - improved haemoglobin
                    - improved cardiovascular stability in first 24 hours
                    - lower transfusion requirements
    - start clock/note time
    - dry, wrap, stimulate, keep warm

        - if preterm (<32 weeks):
            - place undried in plastic wrap and radiant heat
            - inspired oxygen 28-31 weeks 21-30%
                - <28 weeks 30%
            - if giving inflations start with 25cm H_2O
    - assess
        - colour
        - tone
        - breathing
        - heart rate
    - ensure an open airway
        - preterm:
            - consider CPAP
    - if gasping/not breathing:
        - give 5 inflations (30 cm H_2O) – start in air
        - apply PEEP 5-6 cm H_2O if possible
        - apply SpO_2 +/- ECG
    - reassess
        - if no increase in heart rate, look for chest movement
        - if chest not moving:
            - check mask, head and jaw position
            - 2 person support
            - consider suction, laryngeal mask/tracheal tube
            - repeat inflation breaths
            - consider increasing the inflation pressure
    - reassess
        - if no increase in heart rate, look for chest movement
            - once chest is moving, continue ventilation breaths
        - if heart rate not detectable or <60 bpm after 30 seconds of ventilation:
            - synchronise 3 chest compressions to 1 ventilation
            - increase oxygen to 100%
            - consider intubation if not already done or laryngeal mask if not possible
    - reassess heart rate and chest movement every 30 seconds
        - if heart rate remains not detectable or <60 bpm:
            - vascular access and drugs
            - consider other factors, e.g. pneumothorax, hypovolaemia, congenital abnormality
    - update parents and debrief team
    - complete records
- acceptable pre-ductal SpO_2
    - 2 min – 65%
    - 5 min – 85%
    - 10 min – 90%
- advanced resuscitation of the newborn (Resus Council)
    - ABC approach
    - follow newborn life support algorithm

- worrying or potentially life-threatening features?
    - no
        - observe and reassess as necessary
    - yes
        - call for help
        - worrying features
            - reassess ABC
            - consider DEF
            - consider further diagnostic tests and definitive treatments
            - remember thermal care, documentation and debriefing
        - potentially life-threatening features
            - treat potentially life-threatening features and if necessary start CPR
            - continue newborn life support
            - reassess ABCDEF
                - **A**irway
                - **B**reathing
                - **C**irculation
                - **D**isability/**D**rugs/**D**extrose
                - **E**xposure/**E**nvironment
                - **F**amily
            - treat underlying cause
            - remember thermal care, documentation and debriefing

NeoC2 neonatal sepsis (NICE guidelines)

- advice for parents of new babies before discharge
    - seek medical help if their baby:
        - is showing abnormal behaviour (e.g. inconsolable crying or listlessness)
        - is unusually floppy
        - has an abnormal temperature unexplained by environmental factors (<36° or >38°)
        - has abnormal breathing (rapid breathing, difficulty in breathing or grunting)
        - has a change in skin colour (e.g. very pale, blue/grey or dark yellow)
        - has developed new difficulties with feeding
- risk factors for early onset neonatal infection
    - red flag risk factor
        - suspected or confirmed infection in another baby in the case of multiple pregnancy
    - other risk factors
        - invasive group B streptococcal infection in a previous baby or maternal group B streptococcal colonisation, bacteriuria or infection in the current pregnancy
        - pre-term birth following spontaneous labour before 37 weeks' gestation
        - confirmed rupture of membranes for more than 18 hours before a pre-term birth
        - confirmed prelabour rupture of membranes at term for more than 24 hours before the onset of labour

        - intrapartum fever higher than 38° if there is suspected or confirmed bacterial infection
        - clinical diagnosis of chorioamnionitis
- clinical indicators of possible early-onset neonatal infection
    - red flag clinical indicators
        - apnoea
        - seizures
        - need for cardiopulmonary resuscitation
        - need for mechanical ventilation
        - signs of shock
    - other clinical indicators
        - altered behaviour or responsiveness
        - altered muscle tone (e.g. floppiness)
        - feeding difficulties (e.g. feed refusal)
        - feed intolerance
            - including vomiting, excessive gastric aspirates, abdominal distension
        - abnormal heart rate (bradycardia or tachycardia)
        - signs of respiratory distress (e.g. grunting, recession, tachypnoea)
        - hypoxia (e.g. central cyanosis or reduced sats)
        - persistent pulmonary hypertension of newborns
        - jaundice within 24 hours of birth
        - signs of neonatal encephalopathy
        - temperature abnormality (<36° or >38°) unexplained by environmental factors
        - unexplained excessive bleeding, thrombocytopenia or abnormal coagulation
        - altered glucose homeostasis (hypoglycaemia or hyperglycaemia)
        - metabolic acidosis (base deficit of 10 mmol/L or greater)
    - in babies without red flags and only 1 risk factor or 1 clinical indicator, use clinical judgement to decide:
        - whether it is safe to withhold antibiotics
        - whether the baby's vital signs and clinical condition need to be monitored
            - if monitoring is needed, continue for at least 12 hours using a newborn early warning system
- investigations before starting antibiotics in babies who may have early-onset infection
    - blood culture
    - baseline CRP
    - if safe to do so, lumbar puncture if:
        - strong clinical suspicion of early-onset neonatal infection or
        - clinical symptoms or signs suggesting meningitis
    - site-specific infections
        - swabs for purulent eye discharge
        - clinical signs of umbilical infection (e.g. purulent discharge or signs of periumbilical cellulitis):
            - blood culture
            - swab for microscopy and culture
            - IV flucloxacillin and gentamicin
- antibiotics for suspected early-onset infection

    - IV benzylpenicillin with gentamicin as first choice empirical treatment
        - benzylpenicillin 25mg/kg every 12 hours (8 hours if unwell)
        - gentamicin 5mg/kg every 36 hours (shorter interval if baby very unwell or blood culture shows Gram-negative infection)
- risk factors for late-onset neonatal infection
    - prematurity
    - mechanical ventilation
    - history of surgery
    - presence of central catheter
- clinical indicators of possible late-onset neonatal infection
    - behaviour
        - parent or caregiver concern for change in behaviour
        - appears ill to a healthcare professional
        - does not wake, or if roused does not stay awake
        - weak high-pitched or continuous cry
    - respiratory
        - raised respiratory rate (60 breaths per minute or more)
        - grunting
        - apnoea
        - oxygen saturation of <90% in air or increased oxygen requirement over baseline
    - circulation and hydration
        - persistent tachycardia (160bpm or more)
        - persistent bradycardia (<100bpm)
    - skin
        - mottled or ashen appearance
        - cyanosis of skin, lips or tongue
        - non-blanching rash of skin
    - other
        - temperature 38° or more unexplained by environmental factors
        - temperature <36° unexplained by environmental factors
        - alterations in feeding pattern
        - abdominal distension
        - seizures
        - bulging fontanelle
- investigations for late-onset infection
    - as for early onset
    - LP unless contraindicated:
        - infants <1 month
        - infants 1-3 months who appear unwell
        - infants 1-3 months with WCC <5 x10^9/L or >15 x 10^9/L
- management
    - antibiotics
        - if >40 weeks corrected gestational age:
            - ceftriaxone 50mg/kg
        - if ≤40 weeks corrected gestational age
            - cefotaxime 50mg/kg every 6-12 hours

- fluid resuscitation, if required:
    - 10-20mg/kg 0.9% sodium chloride over <10 minutes

NeoC3 cyanotic/non-cyanotic congenital heart disease

- background
    - congenital heart disease is the commonest congenital malformation
    - affects 9 in every 1000 newborns
    - 80-85% survive to adulthood
    - one of four problems:
        - not enough pulmonary blood flow
        - too much pulmonary blood flow
        - not enough systemic blood flow
        - not enough coronary artery blood flow
            - if the child is blue, there is not enough pulmonary blood flow
            - if the child is pink they may have too much pulmonary blood flow
            - if the child is grey, there is not enough systemic flow
- classification
    - cyanotic (caused by right-left shunting where de-oxygenated blood mixes with oxygenated blood)
        - tetralogy of Fallot
            - 10% of cyanotic heart disease
            - most common cause of cyanosis in children over 8 months
            - four elements:
                - ventricular septal defect
                - right ventricular hypertrophy
                - overriding aorta
                - pulmonary stenosis
            - baseline status depends on degree of pulmonary stenosis (or pulmonary outflow obstruction)
                - if mild, there is normal pulmonary blood flow ('pink tets'); they have normal sats and may be much older before diagnosis
                - if there is significant outflow tract obstruction, the patient is hypoxic, usually with sats in the 70s
            - tet spells
                - if systemic vascular resistance drops, the patient's baseline status changes
                    - causes oxygenated blood to be preferentially shunted from the right side of the heart, through the VSD to the left side of the heart (rather than going through the outflow obstruction)
                - a spell can be preceded by an innocuous event (e.g. crying)
                - results in falling pO2, rising CO2 and falling pH
                    - all increase the pulmonary vascular resistance and stimulate the respiratory centre (consequently increasing venous return)

        - increased venous return results in more shunting and a worsening of the cycle
    - management of tet spell
        - increase systemic vascular resistance, decrease venous return or drop the pulmonary vascular resistance
        - best management plan is:
            - knees to chest
            - calm the patient
            - high flow oxygen
            - morphine and IV fluids
            - if still having trouble: ketamine (IM or IV)
- repair
    - staged repair involving Blalock-Taussig shunt (a manmade ductus arteriosus), used until the patient is big enough for a definitive repair
    - definitive repair is normally done by 6 months
- survival is 86% at 30 years
- there is right bundle in the majority of post-op patients; some have pulmonary regurgitation or a recurrent stenosis
- right ventricular dysfunction is common and it is easy to fluid overload
- there is a risk of sudden cardiac death
- arrythmias are also common

- transposition of great vessels
    - occurs due to failure of septation of the pulmonary artery and aorta
    - results in two parallel circulations, where deoxygenated blood is pumped around the body and oxygenated blood around the lungs
        - results in severe cyanosis, metabolic acidosis and death unless there is mixing (e.g. via VSD, ASD, PDA)
    - investigations
        - ECG – usually normal
        - CXR – may show egg on string appearance or signs of CCF
    - management
        - if no VSD, cyanosis occurs when the PDA closes – requires prostaglandin infusion
        - if VSD, patients are usually well-oxygenated but prone to developing CCF – require surgical repair within first few weeks
        - some infants with intact ventricular septum remain excessively cyanotic despite PGE infusion and require balloon atrial septostomy to create ASD
- hypoplastic left heart
    - common diagnosis, usually diagnosed prenatally
    - most patients are initially haemodynamically stable
        - over several days, as pulmonary vascular resistance falls and the ductus arteriosus closes, the ratio of pulmonary to

systemic blood flow increases, causing increased right to left shunting, leading to cyanosis, congestive heart failure, shock and respiratory distress

- examination
    - absent femoral and brachial pulses
    - delayed cap refill
    - hypotension secondary to shock
    - signs of right heart failure
        - enlarged liver
        - tachypnoea
        - rales
    - shock and respiratory distress secondary to acidosis upon ductus arteriosus closure
    - may have PDA murmur
- investigations
    - ECG – absent LV forces
    - CXR – cardiomegaly
    - echo – gold standard for diagnosis
- management
    - patients should be born in a cardiac centre
    - prostaglandin infusion to maintain ductus arteriosus patency
    - surgery within 3-5 days – requires 3 staged operations to establish a single ventricle circulation (Norwood, Glenn, Fontan)

- pulmonary atresia
- Eisenmenger syndrome (later in life, pulmonary hypertension with right to left shunt)
- truncus arteriosus
    - the only artery arising from the heart functions as both the aorta and pulmonary artery
    - mixed blood is pumped to the lungs and body
    - examination
        - wide pulse pressure
        - systolic ejection murmur – increased flow across semi-lunar valve
        - single second heart sound
    - management
        - surgical repair via a Rastelli procedure – involves a patch to close the VSD and allow the LV to communicate with the common trunk; a conduit is then made from the RV to the pulmonary artery
- tricuspid atresia
    - lack of development of the tricuspid valve with no inlet to the RV
    - there is a right to left shunt through the foramen ovale
    - examination
        - harsh systolic murmur

- investigation
    - ECG: superior axis, absent RV voltages, large P waves
    - CXR: may have increased or decreased pulmonary vascular markings
- management
    - if presenting with cyanosis, requires BT shunt or RV to pulmonary artery conduit shortly after birth
    - if presenting with CCF, medical management is used initially followed by surgical correction
    - definitive management is to establish Fontan circulation by 3-5 years of age

- total anomalous pulmonary venous drainage
    - the four pulmonary veins do not drain into the left atrium, but into the innominate vein, liver or cardiac sinus/right atrium
    - presentation
        - depends on degree of obstruction between the pulmonary veins and right heart
        - in supracardiac and infracardiac lesions there is usually a higher degree of obstruction, which presents at birth with pulmonary oedema, pulmonary hypertension and cyanosis (collapsed cyanotic neonate)
        - in cardiac lesions there is less obstruction and symptoms develop within weeks to months with mild cyanosis and CCF
    - investigations
        - ECG: right axis deviation, RVH
        - CXR: snowman sign
        - definitive imaging: echo, cardiac MRI, angiography
    - management
        - if patients present obstructed – emergency surgical correction
        - if non-obstructed – medical management of CCF followed by elective repair
- Ebstein anomaly (inferior displacement of tricuspid valve into right ventricle with right to left shunt through ASD)

- acyanotic
    - left to right shunt
        - ventricular septal defect
            - examination
                - usually asymptomatic and undetectable unless large (larger than aortic valve)
                - may have pansystolic murmur at lower left sternal edge (usually the louder the murmur, the smaller the hole)
            - investigations
                - ECG: biventricular hypertrophy by 2 months
                - CXR: increased pulmonary vascular markings and cardiomegaly

        - management
            - trial of medical management
            - avoid supplemental oxygen and hyperventilation (cause pulmonary vascular resistance to fall causing increased shunting)
            - muscular VSDs often close spontaneously, perimembranous ones do not
                - they are closed surgically or percutaneously (IR procedure)
                - surgical correction is deferred to 3-5 months
    - atrial septal defect
        - rarely cause symptoms in infants if isolated – significant shunting does not occur until right ventricular compliance is less than left, leading to CCF at around 2-3 years
        - examination
            - soft systolic murmur at upper sternal edge
        - investigations
            - ECG: partial RBBB, RV hypertrophy
            - CXR: cardiomegaly, increased pulmonary vascular markings
        - management
            - medical management of symptoms
            - surgical closure at 3-5 years (90% with device closure in catheter lab)
    - atrioventricular septal defect
    - patent ductus arteriosus
        - usually asymptomatic in isolation
        - examination
            - continuous systolic murmur in the left infraclavicular area – known as a machinery murmur
        - investigations
            - ECG: usually normal
            - CXR: increased pulmonary vascular markings if large
        - management
            - two thirds will close with a course of indomethacin or ibuprofen (prostaglandin inhibitors)
            - if large enough to cause CCF – medical management and surgical ligation at 1-3 months
            - if no CCF – closure in cath lab with a coil or device at 1 year
            - slightly increased risk of bacterial endocarditis if left patent
- no shunt
    - bicuspid aortic valve, aortic stenosis
    - pulmonary stenosis
    - tricuspid stenosis
    - coarctation

                - defined as critical when the systemic circulation depends on ductus arteriosus patency
                - can also remain asymptomatic until adulthood
                - often associated with other abnormalities such as VSD or bicuspid aortic valve
                - examination
                    - absent femoral pulses or radio-femoral delay before the ductus arteriosus closes
                    - cyanosis
                    - 4 limb BP discrepancies
                        - gradient of >10mmHg between upper and lower limbs is clinically significant
                    - signs of CCF
                - investigations
                    - diagnosis usually made antenatally on US scan
                    - ECG may show LVH or RV conduction delay
                    - CXR may show figure of 3 sign
                    - all patients will have cardiac MRI or CT for evaluation of thoracic aorta prior to definitive management
                - management
                    - rapid A-E assessment
                    - prostaglandin infusion if duct-dependent circulation
                    - diuretics to reduce preload combined with volume replacement to correct metabolic acidosis
                    - surgery usually performed within 24 hours with removal of the narrow segment
        - dextrocardia
- management of congestive cardiac failure
    - heart is unable to supply blood to meet tissue demands, with heart failure caused by left to right shunting
        - oxygenated blood has a higher pressure so there is increased flow through the pulmonary vasculature, resulting in pulmonary oedema and breathlessness
        - the total volume being pumped into the systemic circulation is lower, resulting in poor tissue perfusion, causing fatigue, poor nutrition and failure to thrive
    - often presents in the first 1-3 months of life
    - presentation in neonates
        - symptoms
            - feeding difficulty
            - sweating/tachypnoea with feeds
            - failure to thrive
            - respiratory distress
            - fussiness
        - signs
            - tachypnoea and laboured breathing
            - rales

                - hepatomegaly
                - cyanosis if severe
                - faltering growth
                - displaced apex beat
                - cool peripheries
                - gallop rhythm
        - medical management
            - loop diuretics
            - digoxin
            - ACEis
            - beta blockers
            - spironolactone

NeoC4 jaundice

- background
    - bilirubin metabolism
        - unconjugated bilirubin (indirect bilirubin) is created from red blood cell breakdown and transported into the bloodstream mostly bound to albumin
        - unconjugated bilirubin is transported into the hepatocytes and conjugated with glucuronic acid
        - conjugated bilirubin (direct bilirubin) is excreted into the biliary system and, ultimately, the small intestine
        - most bilirubin is excreted into the stool, but a small proportion is reabsorbed as part of the entero-hepatic circulation via the portal vein
- reasons for neonates being more prone to jaundice
    - they have a relative polycythaemia and therefore increased cell breakdown
    - the liver is relatively immature and thus unable to cope with normal bilirubin metabolism
    - changes in intestinal flora alter the entero-hepatic circulation
- risk factors for significant neonatal jaundice
    - low birth weight – premature and small for dates
    - breastfed babies
    - previous sibling with neonatal jaundice requiring phototherapy
    - visible jaundice in first 24 hours
    - infants of mothers who have diabetes
    - male infants
    - East Asians
    - populations living at high altitudes
- neonatal jaundice
    - physiological
        - in most babies it is self-limiting and harmless
        - very common, develops in 60% of term and 80% of preterm infants (<37 weeks) in the first few weeks of life
        - develops after the first 24 hours
        - resolves by 14 days of life in term (37—42 weeks) or 21 days in preterm infants
        - child is systemically well and thriving

        - total serum bilirubin is less than the treatment threshold
    - pathological
        - jaundice developing within the first 24 hours
            - likely to be related to a haemolytic process (e.g. ABO incompatibility) and is always pathological
            - in young babies bilirubin can pass the blood-brain barrier and high levels can lead to long-term CNS damage (kernicterus)
            - these patients should have urgent serum levels taken and urgent paediatric referral
        - jaundice not resolving by 14 days of life (term) or 21 days (preterm)
            - prolonged jaundice can be a clue to serious underlying liver disease such as biliary atresia
            - patients require investigation and paediatric referral (following local guidelines)
        - child is unwell or failing to thrive
            - alternative diagnosis should be sought
                - e.g. sepsis, dehydration, inborn errors of metabolism
            - it is normal for the baby to lose up to 10% of their birth weight
                - this should be regained by 2 weeks of life
            - if jaundice is diagnosed in the context of a baby who has lost significant weight, dehydration should be considered
        - total serum bilirubin is greater than the treatment line
            - NICE guidelines have treatment thresholds based upon gestation at birth and hours of life
                - serum levels should be compared to the graph and treatment started as appropriate
- management
    - if there is no evidence of pathological jaundice, the child can be discharged home with advice to return if unwell or if the jaundice is increasing
    - if the measured bilirubin level is above the threshold, the child should be referred for phototherapy and investigation
        - threshold graphs have a treatment line for phototherapy and another for exchange transfusion
            - paediatric units often favour intensive phototherapy unless there is evidence of kernicterus
            - treatment levels are based on expert opinion and there is no evidence base
        - phototherapy
            - involves placing the child under a lamp emitting light in the blue spectrum
                - this converts bilirubin in the skin to a form that can be excreted in the urine
            - phototherapy has been shown to significantly reduce hyperbilrubinaemia when compared to no treatment in both term and preterm babies

NeoC5 feeding patterns

- medical advantages of breastfeeding
    - child
        - immunity/infection protection (including reduced hospital admissions for infection)
        - protective effect on more severe eczema
        - may be protective against childhood asthma
        - associated with a reduction in sudden infant death syndrome
        - positive effect on intelligence
        - may protect against later risks of overweight and obesity
        - protects against type 2 diabetes and possibly type 1
    - mother
        - decreased risk of invasive breast cancer
        - reduction in risk of type 2 diabetes
        - reduction in risk of ovarian cancer with longer periods of breastfeeding
        - possible reduced risk in metabolic syndrome and cardiovascular disease
        - contraceptive effect if:
            - child is up to 6 months old
            - mother is amenorrhoeic
            - child is exclusively breastfed
- issues to be aware of with breastfeeding
    - vitamin D
        - breast milk is low in vitamin D
        - infants may require supplementation with drops from 1 month of age if mum has not taken vitamin D supplementation throughout pregnancy
    - HIV
        - can be transmitted via breastfeeding
        - UK guidance is that mothers who are HIV positive, regardless of maternal viral load and antiretroviral therapy do not breastfeed from birth
            - however it may be the safer option if moving to a country where safe infant formula is unavailable
    - hepatitis B and C
        - breastfeeding is not a risk factor for hepatitis B transmission if the infant has received appropriate HBV immunoprophylaxis
        - HCV can be found in breast milk but breastfeeding is not contraindicated
    - other infections
        - breastfeeding should be stopped for 24 hours for:
            - Neisseria gonorrhoeae
            - Haemophilus influenzae
            - group B streptococci
            - staphylococci
        - longer for other infections:
            - Borrelia burgdorferi
            - Treponema pallidum
            - Mycobacterium tuberculosis
- problems for mum
    - cracked/sore nipples

        - very common during the first weeks of breastfeeding
        - may be caused by:
            - improper position of baby
                - altering feeding positions may help to reduce soreness
            - improper feeding technique
                - may be caused by incomplete suction release at the end of baby's feeding
                - gently inserting a finger into the side of the mouth to break the suction may help
            - improper nipple care
                - excessively dry or excessively moist skin can cause soreness
                - moisture can be caused by synthetic bras
                - ointments containing lanolin may be helpful
                - olive oil and expressed milk may also be useful
                - regardless of treatment, for most women initial nipple pain reduces to mild levels after 7-10 days postpartum
    - blocked duct and breast engorgement
        - caused by poor drainage of the breast
        - the breasts feel swollen, hard and painful; there may be redness or systemic symptoms; the nipples cannot protrude to allow the baby to 'latch on' and feeding becomes difficult
        - common causes are:
            - pressure on the breast (e.g. from poorly fitting bra or seatbelt)
            - prolonged gaps between feeds
        - management
            - nurse 8 times or more in 24 hours for at least 15 minutes for each feed to prevent engorgement
            - to manage it: express milk manually or with a pump; alternating warm showers with cold compresses can help with the discomfort
        - if engorgement persists, mastitis may develop and milk or milk products can get into the bloodstream leading to flu-like symptoms similar to those of incompatible blood transfusion
    - mastitis/abscess
        - mastitis occurs in 20% of breastfeeding women
        - risk is increased with:
            - nipple damage
            - over-supply of milk
            - use of nipple shields
            - nipple carriage of Staph. aureus
        - may be infective or non-infective
        - treatment may include antibiotics (e.g. flucloxacillin) if symptoms have not resolved within 24 hours
            - an abscess may require aspiration or incision and drainage
        - breastfeeding (or pumping to relieve breast engorgement) should continue whilst receiving treatment
    - insufficient milk/hungry baby
        - most common reason for giving up breastfeeding

            - there is usually adequate milk but the woman may not be aware that her breasts will soften as feeding becomes established and that it is normal for some breastfed babies to feed as often as 10 times a day
            - frequent feedings, offering both breasts at each feed, adequate rest, good nutrition and adequate fluid intake can help maintain a good milk supply
            - expressing after feeds will increase milk supply
            - checking weight and growth will determine whether the baby is taking enough milk
- establishing breastfeeding
    - WHO recommendations:
        - breastfeeding should begin within an hour of birth
        - breastfeeding should be 'on demand', as often as the child wants, day and night
        - bottles or pacifiers should be avoided if possible (evidence of negative effect is lacking)
    - mothers need support, confidence and encouragement for successful breastfeeding, including immediate breastfeeding support at delivery
- formula feeding
    - infant formula is the only alternative to breast milk
    - cows' milk is not suitable until the baby is 1 year old as it contains too much salt and protein and not enough iron
    - follow-on milks can be given from 6 months, although this is not normally necessary
    - hydrolysed protein infant formulas can be prescribed if the baby has an allergy to cows' milk
    - soya-based infant formulas can be used
- introducing solids/weaning
    - there is still debate around optimal time for weaning
    - the WHO recommends exclusive breastfeeding for 6 months
        - they recommend introduction of solids at 6 months (26 weeks) whilst continuing to breastfeed
        - in the UK, Department of Health guidelines recommend the introduction of solid food 'at around six months'
    - the British Dietetic Association recommends:
        - exclusive breastfeeding from birth, until the introduction of solid foods, is optimal
        - breastfeeding should continue throughout complementary feeding
        - introduction of solid food should start at around 6 months but development varies widely and some babies may be ready before or after this time
        - the introduction of solid food should start no later than 6 months (26 weeks) and not before 4 months (17 weeks)
        - preterm infants require advice from the dietician and medical team caring for them
- weight loss in baby
    - it is normal for newborns to lose up to 10% of their birthweight, but this should be regained at around day 10
    - %weight loss = (birthweight – current weight)/birthweight x100
    - those who have lost more than 10% usually require admission
- feeding history

- general
    - is baby bottle or breast fed or a mixture (what proportion bottle/breast)?
    - has the type of feed changed since birth?
    - does baby wake spontaneously for feeds or cry for feeds or do the parents have to wake the baby?
    - is the baby vomiting (large amounts) after feeds or just small spit-ups?
        - what colour is the vomit?
            - if green, surgical review required
    - are they burping/winding the baby after feeding? how long does this take?
    - how many wet nappies and how many dirty nappies per day?
    - what colour is the urine?
        - red/orange 'brick dust' coloured staining in the nappy can be due to the presence of urate crystals, a sign of dehydration
    - has baby passed any stool since birth?
        - what colour are the stools?
            - black sticky meconium is normal initially, becoming lighter, greenish-yellow to bright yellow and looser ('seedy') in texture
- bottle feeding
    - how much is baby taking per feed?
        - millilitres or ounces
        - 1oz = 30ml
    - how many hours between each feed?
    - how long does it take baby to finish a feed?
    - what type of formula are they using and have they changed this recently?
- breast feeding
    - has mum's milk 'come in' yet?
        - if so, mum will notice nipples leaking or may be able to hand express some milk from the nipple
        - is it colostrum (thin clear yellow liquid) or milk?
    - is this the first baby?
        - milk can take up to 3 days to come in, usually quicker if not the first baby
    - how many hours between each feed?
    - how long does baby stay on the breast for?
    - is baby alternating between breasts?
    - is it painful when baby latches on, and if so has the midwife given any advice on this yet?
    - does baby seem satisfied after feeding?
        - if well-fed will usually sleep for several hours after a feed
    - do mum's breasts feel different/less full after a feed?

- examination of baby
    - general physical examination
    - check with finger and pen torch for cleft palate
    - whether the skin on the abdomen appears loose or wrinkly – suggests significant weight loss
    - look in the nappy

        - wet?
        - urate crystals ('brick dust')? – usually a sign baby is slightly underfed
    - blood glucose (heelprick BM)
    - jaundice (sclerae/palate) – dehydrated babies are often jaundiced and vice versa
    - check the suck reflex with gloved little finger
    - whether there is a patent anus
- how much milk baby should take
    - all babies are different and some are hungrier than others
    - they need gradually increasing amounts over the first few days
        - first day of life – 60ml/kg/day
        - second day of life – 75ml/kg/day
        - third to fourth day of life – 90ml/kg/day
        - fourth to fifth day of life – 120ml/kg/day
        - by seventh day of life – 150ml/kg/day
    - if mum is breastfeeding and concerned about quantities, she could potentially hand express or use a breast pump to have an idea of how much milk she is producing
- safety netting for discharge
    - return if baby:
        - is not feeding at all
        - is not passing urine at all
        - is vomiting large amounts or vomit is green
        - is lethargic or floppy
        - has a fever
        - has a rash

NEPHROLOGY

NepP1 electrolyte disorders

- potassium
    - hyperkalaemia
        - intake
            - oral intake
            - blood transfusion
        - redistribution
            - acidosis
            - rhabdomyolysis
            - tumour lysis
        - output
            - urinary
                - renal tubular acidosis type 4
                - renal failure
                - adrenal insufficiency
                - diabetes
                - potassium-sparing diuretics
    - hypokalaemia
        - intake
            - inadequate intake
        - redistribution

                - alkalosis
                - hypomagnesaemia
                - glucose infusion
                - periodic paralysis
                - beta-agonists
            - output
                - urinary
                    - steroids (exogenous or endogenous)
                    - DKA
                    - hyperaldosteronism
                    - Cushing's
                    - renal tubular acidosis
                    - diuretics
                - non-urinary
                    - upper GI
                        - vomiting
                    - mid GI
                        - fistula
                    - lower GI
                        - diarrhoea
                    - other
                        - sweat
                        - burns
                        - bleeding
                        - renal replacement therapy
- magnesium
    - hypermagnesaemia
        - intake
            - usually iatrogenic (magnesium infusion)
        - output
            - renal failure increases risk of accumulation
    - hypomagnesaemia
        - intake
            - TPN
            - malabsorption
            - alcoholism
        - redistribution
            - insulin
            - hungry bone syndrome
        - output
            - urinary
                - renal tubular acidosis
                - diuretics
                - polyuria from any cause
            - non-urinary
                - upper GI
                    - NG loss

        - lower GI
            - diarrhoea
- calcium
    - hypercalcaemia
        - intake
            - calcium
            - vitamin A or D
            - hypomagnesaemia
            - hypovolaemia
            - TPN
        - redistribution
            - immobilisation
            - malignancy
            - hyperparathyroidism
            - sarcoid
            - lithium
            - adrenal insufficiency
            - endocrine causes (thyrotoxicosis, acromegaly, phaeochromocytoma)
        - output
            - urinary
                - thiazides
    - hypocalcaemia
        - intake
            - calcium
            - vitamin D
            - phenytoin (increased metabolism of vitamin D0
        - redistribution
            - alkalosis
            - citrate toxicity
            - hyperphosphataemia
            - pancreatitis
            - tumour lysis syndrome
            - rhabdomyolysis
            - decreased bone turnover
            - hypoparathyroidism
            - drugs (bisphosphonates, PPIs, SSRIs, gentamycin)
        - output
            - urinary
                - ethylene glycol
                - cisplatin
                - protamine
                - loop diuretics
            - non-urinary
                - other
                    - bleeding
                    - plasmapheresis
                    - citrate renal replacement therapy

- phosphate
    - hyperphosphataemia
        - output
            - non-urinary
                - other
                    - skin
                    - bleeding
                    - sweat
                    - renal replacement therapy
    - hypophosphataemia
        - intake
            - malnutrition
            - phosphate binders
            - vitamin D
            - malabsorption
            - TPN
        - redistribution
            - refeeding syndrome
            - insulin in DKA
        - output
            - urinary
                - diuretics
                - osmotic diuresis
                - hyperparathyroidism
                - proximal tubular dysfunction (Fanconi syndrome)
            - non-urinary
                - lower GI
                    - diarrhoea
                - other
                    - sweat
                    - burns
                    - sepsis
                    - bleeding
- sodium
    - hypernatraemia
        - intake
            - high osmolality rehydration solutions
            - salt poisoning
            - inappropriate formula concentration
            - iatrogenic (hypertonic saline, sodium bicarbonate)
        - redistribution
            - hyperaldosteronism
            - CCF
            - nephrotic syndrome
        - output
            - urinary
                - osmotic diuretics

        - diabetes
        - acute tubular necrosis
    - non-urinary
        - lower GI
            - diarrhoea
            - stomal losses
        - other
            - sweat
            - burns

- hyponatraemia
    - hypotonic
        - hypovolaemic
            - urinary (high urinary sodium)
                - diuretics
                - osmotic diuretics
                - renal tubular acidosis
                - salt wasting
                - mineralocorticoid deficiencies
            - non-urinary (low urinary sodium)
                - upper GI
                    - vomiting
                - mid GI
                    - pancreatitis
                    - bowel obstruction
                - lower GI
                    - diarrhoea
                    - bowel preparation
                - other
                    - skin
                    - bleeding
                    - sweat
        - euvolaemic
            - SIADH (most common)
            - psychogenic polydipsia
            - hypotonic IV fluid therapy
            - adrenal insufficiency
            - hypothyroidism
        - hypervolaemic
            - CHF
            - cirrhosis
            - nephrotic syndrome
            - hypothyroidism
            - pregnancy
            - TURP syndrome
    - isotonic (pseudohyponatraemia)
        - high proteins
        - high lipids

- hypertonic
    - glucose
    - mannitol
    - sorbitol
    - radiocontrast
    - advanced renal disease

NepP2 oliguria

- definition
    - urine output:
        - <1ml/kg/hr in infants
        - <0.5ml/kg/hr in children
        - <400ml/day in adults
- aetiology
    - pre-renal (most common), renal or post-renal
    - risk increased in intensive care and surgical patients
    - rarely found in CKD
    - pre-renal causes include:
        - dehydration
        - vascular collapse
        - low cardiac output
    - renal causes
        - structural renal damage, e.g. acute tubular necrosis, primary glomerular diseases, vascular lesions
    - post-renal causes
        - mechanical or functional obstruction to the flow of urine
        - most common cause is blocked catheter
        - usually responds to release of the obstruction
- history
    - excessive fluid loss
        - diarrhoea, vomiting
    - drug use
        - e.g. gentamicin, NSAIDs
    - gross haematuria and oedema in children suggesting glomerular disease
    - symptoms of urinary tract obstruction
        - e.g. complete failure to pass urine
- examination
    - patient may be unwell (breathless, clammy, peripherally shut down with unrecordable blood pressure)
    - may be signs due to AKI – oedema, anaemia
    - signs of congestive heart failure
    - hypertension
    - signs of underlying disease
- investigations
    - urine midstream dipstick and microscopy
        - prerenal
            - few hyaline casts and fine granular casts

                - little protein, haemoglobin or red cells
            - intrinsic
                - haematuria and proteinuria are prominent
                - broad brown granular casts in ischaemic or toxic acute tubular necrosis
                - red cell casts usually found in acute glomerulonephritis
                - white cells in acute interstitial nephritis
    - urinary electrolytes
        - valuable indicators of functioning renal tubules
    - bloods
        - consider CRP
        - U&Es
        - FBC – anaemia from dilution and decreased erythropoiesis
        - blood gas for acid base status
    - renal doppler ultrasound
    - renal biopsy may be required later if pre-renal and post-renal causes have been ruled out
- management
    - treatment of any reversible causes
    - restoration of intravascular volume
    - strict fluid balance
    - correction of electrolyte abnormality
    - daily weights
    - dialysis/filtration if requirements met
        - indications may include:
            - volume expansion that cannot be managed with diuretics
            - hyperkalaemia refractory to medical therapy
            - uraemia
    - surgical
        - e.g. nephrostomy
            - urinary catheter passed percutaneously under local anaesthetic into the renal pelvis and connected to a drainage bag
- complications
    - cardiovascular
        - hypertension
        - congestive heart failure
        - pulmonary oedema
        - arrhythmia secondary to hyperkalaemia
    - gastrointestinal
        - anorexia
        - nausea
        - vomiting
        - ileus
    - haematological
        - anaemia
        - platelet dysfunction
    - infections

- neurological
    - confusion
    - sleepiness
    - seizures

NepC1 acute kidney injury

- background
    - rapid reduction in kidney function resulting in a failure to maintain fluid, electrolyte and acid-base homeostasis
    - most commonly staged using the Acute Kidney Injury Network staging criteria
        - initially developed as a research tool but allows risk stratification
        - less useful in the ED where serum creatinine and urine output may not be available
    - NICE recommendations for AKI detection (in line with pRIFLE, KDIGO and AKIN criteria):
        - rise in serum creatinine of 26+ μmol/L in 48 hours
        - 50% or greater rise in serum creatinine in the preceding 7 days
        - drop in urine output to 0.5ml/kg/hr for 6 hours in adults or 8 hours in children and young adults
        - in children and young people a fall in eGFR of 25% or more in the preceding 7 days
- causes
    - pre-renal (90%)
        - volume depletion
            - haemorrhage
            - severe vomiting or diarrhoea
            - burns
            - inappropriate diuresis
        - oedematous states
            - cardiac failure
            - cirrhosis
            - nephrotic syndrome
        - hypotension
            - cardiogenic shock
            - sepsis
            - anaphylaxis
        - cardiovascular
            - severe cardiac failure
            - arrhythmias
        - renal hypoperfusion
            - NSAIDs or COX-2 inhibitors
            - ACE inhibitors of ARBs
            - AAA
            - renal artery stenosis or occlusion
            - hepatorenal syndrome
    - renal (intrinsic)
        - glomerular disease

                - glomerulonephritis
                - thrombosis
                - haemolytic uraemic syndrome
            - tubular injury
                - acute tubular necrosis following prolonged ischaemia
                - nephrotoxins
                    - e.g. aminoglycosides, radiocontrast media, myoglobin, cisplatin, heavy metals, light chains in myeloma
            - acute interstitial nephritis due to drugs (e.g. NSAIDs), infection or autoimmune diseases
            - vascular disease
                - vasculitis
                - cryoglobulinaemia
                - polyarteritis nodosa
                - thrombocytic microangiography
                - cholesterol emboli
                - renal artery stenosis
                - renal vein thrombosis
                - malignant hypertension
            - eclampsia
    - post renal
        - calculus
        - blood clot
        - papillary necrosis
        - urethral stricture
        - prostatic hypertrophy or malignancy
        - bladder tumour
        - radiation fibrosis
        - pelvic malignancy
        - retroperitoneal fibrosis
- risk factors in people with acute illness
    - age ≥65
    - CKD, particularly if eGFR <60
    - past history of AKI
    - co-existing illness (e.g. cardiac failure, liver disease, diabetes)
    - neurological impairment of disability, particularly where reliance on a carer may reduce access to fluids
    - hypovolaemia
    - symptoms or history of urological obstruction or risk factors for this
    - sepsis
    - use of iodinated contrast agents within the previous week
        - increased risk with:
            - CKD (eGFR <40)
            - diabetes if co-existent with CKD
            - heart failure
            - age ≥75
            - hypovolaemia

                - increasing volume of contrast agent
                - intra-arterial administration of contrast agent
        - current or recent medication with nephrotoxic potential
        - deteriorating early warning scores
        - patients in the perioperative period
- symptoms
    - urine output
        - usually oliguria or anuria
            - polyuria may occur due to either reduced fluid reabsorption by damaged renal tubules or the osmotic effect of accumulated metabolites
        - abrupt anuria suggests an acute obstruction, acute and severe glomerulonephritis or acute renal artery occlusion
        - gradual reduction in urine output may indicate a urethral stricture or bladder outlet obstruction (e.g. benign prostatic hyperplasia)
    - nausea, vomiting
    - dehydration
    - confusion
- signs
    - hypertension
    - large painless bladder on abdominal examination typical of chronic urinary retention
    - dehydration with postural hypotension and no oedema
    - fluid overload with raised JVP, pulmonary oedema and peripheral oedema
    - pallor, rash, bruising
        - petechiae, purpura and nosebleed may suggest inflammatory or vascular disease, emboli or DIC
    - pericardial rub
- history
    - drugs
        - nephrotoxic drugs, including over the counter, herbal and recreational
    - occupational/recreational history
        - exposure to sewer systems, tropical diseases, rodents
    - urinary symptoms
    - past medical history
- investigations
    - urine
        - urinalysis
            - mandatory in all patients with AKI
            - if there is blood or protein with no evidence of urinary infection, trauma (e.g. due to catheterisation) and no obvious cause for AKI, consider acute nephritis and refer to nephrologist
        - urine osmolality
    - bloods
        - as guided by history and examination
        - FBC, blood film
            - eosinophilia may be present in acute interstitial nephritis, cholesterol embolization, vasculitis

                - thrombocytopaenia and red cell fragments suggest thrombotic microangiopathy
            - U&Es and creatinine
            - coagulation
                - DIC associated with sepsis
            - CK, myoglobinuria
                - suggestive of rhabdomyolysis
            - CRP
            - immunology
                - serum immunoglobulins, serum protein electrophoresis, Bence Jones proteinuria
                    - myeloma
                - ANA, anti-dsDNA antibodies, ANCA and other autoantibodies
                    - SLE and other autoimmune conditions
                - complement concentrations
                    - low in SLE, acute postinfectious glomerulonephritis, cryoglobulinaemia
                - antiglomerular basement membrane (anti-GBM) antibodies
                    - present in Goodpasture's syndrome
                - antistreptolysin O and anti-DNAase B titres
                    - high after streptococcal infection
            - virology
                - hepatitis B and C
                - HIV
                - important implications for infection control within the dialysis area
        - ultrasound
            - where obstruction is suspected or no cause has been identified
        - other radiology (where appropriate)
            - CXR
                - pulmonary oedema
            - AXR
                - if renal calculi suspected
            - contrast studies
                - e.g. intravenous urogram and renal angiography
                - should be avoided because of the risk of contrast nephropathy
            - Doppler ultrasound
                - renal artery and veins – assessment of possible occlusion
            - magnetic resonance angiography
                - for more accurate assessment of renal vascular occlusion
- differential
    - CKD
        - suggested by:
            - long duration of symptoms
            - nocturia
            - absence of acute illness
            - anaemia
            - hyperphosphataemia, hypocalcaemia

        - similar findings complicate AKI
    - reduced renal size and cortical thickness on renal ultrasound
        - renal size is typically preserved in patients with diabetes
  - acute on chronic kidney disease
- management
  - stop nephrotoxic drugs where possible
  - monitoring
    - creatinine
    - sodium
      - may require oral restriction
    - potassium
      - may require oral restriction
    - calcium
    - phosphate
    - glucose
      - hyperglycaemia may require insulin therapy (which may also help control potassium levels)
      - there is also a risk of hypoglycaemia
  - identify and treat infection
  - optimise fluid balance
    - restore effective circulating volume and avoid overload
    - accurate measurement of urine output
  - urgent relief of urinary tract obstruction
    - e.g. from renal calculi, papillary necrosis, tumours, strictures, prostatic enlargement
  - referral to nephrology for treatment of intrinsic renal disease where appropriate
  - other management of specific problems:
    - angioplasty or stenting of acute renal artery thrombosis
    - consideration of alkaline diuresis for rhabdomyolysis with myoglobinuria
    - consideration of high dose steroids for acute tubulo-interstitial nephritis
    - prednisolone and cyclophosphamide +/- plasma exchange for AKI due to crescenteric glomerulonephritis
    - plasma exchange with fresh frozen plasma for haemolytic uraemic syndrome
  - identify and treat complications
    - including:
      - hyperkalaemia
      - acidosis
      - pulmonary oedema
      - bleeding
  - referral for renal replacement therapy
    - indications (when not responding to medical management) include:
      - hyperkalaemia (>6.5 mmol/L)
      - pulmonary oedema
      - severe metabolic acidaemia (pH ≤7.2) due to kidney failure
      - progressive renal failure (creatinine ≥300 and/or rise in creatinine of >100 μmol/L/day)

                - uraemic complications (pericarditis, encephalopathy)
                - renal transplant
                - CKD (stage 4 or 5)
                - patients suspected of intrinsic renal disease
        - referral to a nephrologist
            - indications include:
                - meeting the criteria for renal replacement therapy
                - uncertainty about cause, management or prognosis
                - where there is a likely diagnosis that will need specialist treatment (glomerulonephritis, vasculitis, tubulo-interstitial nephritis, myeloma)
                - inadequate response to treatment
                - complications
                - history of renal transplant or CKD stage 4 or 5
                - stage 3 AKI according to pRIFLE, AKIN or KDIGO
- prognosis
    - inpatient mortality varies depending on severity, setting and patient factors
        - may be 20-30% in the UK
    - indicators of poor prognosis include:
        - older age
        - multiple organ failure
        - oliguria
        - hypotension
        - number of transfusions
        - acute on chronic kidney disease
    - risk of mortality increases with stage of AKI
    - patients who have had AKI are at greater risk of developing CKD

NepC2 drugs and the kidney

- background
    - the renal interstitium is the area of the kidney between the nephrons
    - most cells are fibroblasts
        - these have various endocrine functions such as production of erythropoietin and prostaglandins
    - any molecule that passes from the tubules to the blood or vice versa has to cross the interstitium
    - there are two main forms of pathology, acute interstitial nephritis and chronic tubulo-interstitial nephritis
- aetiology
    - drug hypersensitivity reaction (most common cause, 40-60%)
        - most frequent examples include:
            - methicillin and other penicillins
            - cephalosporins
            - co-trimoxazole and other sulphonamides
            - rifampicin
            - ciprofloxacin
            - erythromycin

- vancomycin
- all NSAIDs
- diuretics – thiazides, furosemide
- glucosamine

- acute interstitial nephritis
  - background
    - accounts for 6.5-15% of AKI patients
    - almost all caused by hypersensitivity to drugs and not mediated by direct toxicity
  - aetiology
    - beta lactams
      - characteristic clinical picture
      - 2-60 days after treatment starts
      - fever, maculopapular skin rash, hepatic involvement, haematuria
      - renal failure requiring dialysis in around 30% of cases
    - NSAIDs
      - usually in the elderly who have taken it intermittently
      - renal failure several months to years after starting treatment, other signs of drug sensitivity usually missing
      - abundant proteinuria and nephrotic syndrome in most cases
      - renal biopsy shows interstitial changes and diffuse podocyte foot-process fusion with minimal glomerular changes
    - other causes of AIN
      - infection
        - mainly in children following septicaemia
        - presents as acute pyelonephritis with renal micro-abscesses on biopsy
        - also viral infections e.g.
          - Epstein Barr
          - haemorrhagic fevers due to hantaviruses
          - HIV
      - immune disorders
        - e.g. SLE, Sjögren's syndrome, sarcoidosis
      - idiopathic
  - presenting features
    - sudden onset acute kidney injury
      - usually within days of exposure to the causative drug
    - rash
    - fever
    - eosinophilia
    - elevated IgE levels
  - investigations
    - mild to moderate proteinuria
    - nephrotic syndrome only in AIN due to NSAID
    - haematuria only with beta lactam hypersensitivity
    - eosinophilia and eosinophiluria suggests adverse drug reaction
    - kidney normal size or enlarged

        - ultrasound shows increased cortical echogenicity
        - firm diagnosis only possible by renal biopsy
            - shows mononuclear cell infiltration in the interstitium with interstitial oedema
    - management
        - mainly withdrawal of responsible drugs
        - high dose prednisolone may give a faster and more complete reduction in creatinine
- chronic tubulointerstitial nephritis
    - background
        - analgesic nephropathy causes renal papillary necrosis and chronic interstitial nephritis resulting from long term use - usually aspirin and paracetamol
        - renal damage is most apparent in the medulla, starting as prominent thickening of the vasa recta capillaries with patchy areas of tubular necrosis
            - this is followed by papillary necrosis and secondary injury to the cortex with focal and segmental glomerulosclerosis, interstitial infiltration and fibrosis
        - mechanism is unknown but thought to be related to metabolism of paracetamol to reactive intermediates that accumulate in the renal medulla at high concentrations at the papillary tip where they are able to damage the cells lining the duct by oxidation
            - aspirin exacerbates this by depleting glutathione that would detoxify the reactive intermediates and reducing renal blood flow by inhibiting prostaglandins
    - other causes
        - 5-aminosalicylic acid (5-ASA)
            - chemically related to aspirin and has a similar effect
            - inflammation of the interstitium continues for several months or years after taking the drug
            - male: female ratio is 2:15
            - seen during the first year of treatment in a significant proportion
            - usually asymptomatic – patients treated with 5-ASA for inflammatory bowel disease require creatinine monitoring
        - Chinese herbs
            - subacute interstitial nephritis attributed to aristolochic acid
            - main feature is extensive interstitial fibrosis with atrophy and tubular loss, mainly in the cortex
            - there is proteinuria
            - if disease is more severe, progressive renal failure may follow even if herbs are excluded in future
            - steroids may slow the rate of loss of renal function
        - lithium
            - may be implicated in long term use, but not confirmed
        - radiation
            - may occur 6-12 months after radiotherapy with hypertension, anaemia and oedema
            - 50% progress to CKD

            - may alternatively develop over several years, sometimes presenting with hypertension and proteinuria
        - ciclosporin
            - can cause acute and chronic nephrotoxicity
        - lead
            - only becomes evident years after exposure
            - nearly always associated with gout and hypertension
        - cadmium
            - highly toxic metal with the kidney as one of its most important target organs
            - high levels are highly toxic but chronic low level exposure is associated with signs of early renal dysfunction
            - no evidence of progression to CKD
        - metabolic disorders
            - chronic hypokalaemia
            - hyperoxaluria
            - hypercalcaemia
            - chronic urate nephropathy
        - neoplasia
            - myeloma, amyloidosis or leukaemia
        - immunological causes
            - SLE, Sjögren's sarcoidosis
            - vasculitides
        - genetic causes
            - medullary cystic disease
            - Alport's syndrome
    - presentation
        - commonly hypertension, anaemia and proteinuria
        - mostly no urinary tract symptoms but may have flank pain and haematuria
        - diagnosis suggested by:
            - patient 30-70 years
            - long term analgesic use, e.g. for headaches, low back pain, myalgia, weakness
            - may also have a history or symptoms of peptic ulcer disease
    - investigations
        - FBC
        - U&E
        - urinalysis
        - ultrasound
            - may identify hydronephrosis in obstructive pathology
        - CT for greater resolution
    - management
        - depends on aetiology
            - generally supportive treatment, e.g. correction of anaemia
            - in lead nephropathy, chelation is unproven in chronic renal problems but must be implemented in acute poisoning
    - prognosis

        - renal function stabilises or improves slightly on discontinuation of analgesics in early cases
        - may continue to progress in advanced cases, can lead to end stage kidney disease
        - 8-10% develop urinary tract malignancy
            - commonest cause of bladder cancer in women under 50
- examples of nephrotoxic drugs
    - prerenal
        - drugs that cause excessive gastrointestinal losses through diarrhoea or vomiting can cause volume depletion and precipitate AKI
        - NSAIDs, even in short courses, due to renal hypoperfusion
        - ACEi – only a problem in patients with compromised renal perfusion, particularly renal artery stenosis
    - intrarenal
        - penicillamine
        - gold
        - captopril
        - phenytoin
        - some antibiotics
            - penicillins, sulphonamides, rifampicin, cephalosporins
        - furosemide
        - thiazide diuretics
        - NSAIDs
        - drugs causing direct toxicity to renal tubules
            - aminoglycosides
            - amphotericin
            - ciclosporin
    - postrenal (urinary tract obstruction)
        - high dose sulphonamides, acetazolamide. methotrexate – causing crystalluria
        - anticholinergics and alcohol – due to urinary retention
    - others
        - cephalosporins
        - analgesics
        - lithium

NepC3 electrolyte disorders

- general principles
    - intake
    - redistribution
    - output
- potassium
    - hyperkalaemia
        - intake
            - oral intake
            - blood transfusions
        - redistribution

- acidosis
- rhabdomyolysis
- tumour lysis

- output
  - renal tubular acidosis type 4
  - renal failure
  - adrenal insufficiency
  - diabetes mellitus
  - potassium sparing diuretics
- ECG changes
  - peaked T waves
  - prolongation of PR interval
  - widening of QRS
  - reduced or absent P wave
  - AV dissociation
  - sine wave pattern
  - asystole
- management
  - if arrythmia or conduction abnormality, stabilise the myocardium
    - calcium gluconate 10% 10-20ml over 5 minutes
  - shift potassium back into cells
    - 10 units of short acting insulin in 50ml 50% dextrose
      - works in around 15 minutes, lasts for 60 minutes
    - consider giving 10-20ml nebulised salbutamol
      - works in 15-30 minutes, lasts for 2 hours

- hypokalaemia
  - intake
    - inadequate intake
  - redistribution
    - alkalosis
    - hypomagnesaemia
    - glucose infusion
    - periodic paralysis
    - beta-agonists
  - output
    - urinary
      - steroids
        - exogenous or endogenous
      - DKA
      - hyperaldosteronism
      - Cushing's
      - renal tubular acidosis
      - diuretics
    - non-urinary
      - upper GI – vomiting
      - mid GI – fistula
      - lower GI – diarrhoea

        - other
            - sweat
            - burns
            - bleeding
            - renal replacement therapy
    - symptoms
        - generalised weakness and muscle pain
        - constipation
        - paralysis
        - respiratory failure
        - ileus
        - paraesthesia
        - tetany
    - ECG
        - flat T waves
        - ST depression
        - prominent U waves
    - management
        - correct other electrolyte abnormalities such as hypomagnesaemia
        - replace in sodium chloride, maximum rate 20mmol/hour
        - oral supplements (Kay-Cee-L or Sando K) can be used for doses of 40-120 mmol/day
            - including in very low levels if patients are able to take oral supplements, levels are likely to be corrected quickly enough and they have a working GI tract for absorption
- magnesium
    - hypermagnesaemia
        - intake
            - usually iatrogenic
        - redistribution
            - none
        - output
            - urinary
                - renal failure increases risk of accumulation
        - features
            - nausea and vomiting
            - facial flushing
            - hypotension
            - paralytic ileus (due to smooth muscle paralysis)
            - weakness, followed by flaccid muscle paralysis
            - loss of deep tendon reflexes
            - respiratory depression
            - bradycardia
            - complete heart block or cardiac arrest (levels >6.0 mmol/L)
        - management
            - usually withdrawal of the cause is sufficient
            - IV calcium can be used in intensive care with cardiac monitoring

- magnesium loss can be enhanced with IV sodium chloride and furosemide diuresis if urine output is normal
- dialysis may occasionally be required

- hypomagnesaemia
  - intake
    - TPN
    - malabsorption
    - alcoholism
  - redistribution
    - insulin
    - hungry bone syndrome
  - output
    - urinary
      - renal tubular acidosis
      - diuretics
      - polyuria from any cause
    - non-urinary
      - upper GI – NG loss
      - lower GI – diarrhoea
  - symptoms
    - usually asymptomatic until <0.5mmol/L
    - neuromuscular
      - weakness
      - tremor
      - paraesthesia
      - tetany
      - fasciculations
      - seizures, drowsiness, confusion and coma if very low levels
    - cardiovascular
      - arrythmias
  - ECG
    - wide QRS
    - prolonged QT interval
    - flattened T waves
    - U waves
  - management
    - oral or IV replacement
      - IV should not exceed 2g/hour

- calcium
  - hypercalcaemia
    - intake
      - calcium
      - vitamin A or D
      - hypomagnesaemia
      - hypovolaemia
      - TPN
    - redistribution

                - immobilisation
                - malignancy
                - hyperparathyroidism
                - sarcoid
                - lithium
                - adrenal insufficiency
                - endocrine causes
                    - thyrotoxicosis
                    - acromegaly
                    - phaeochromocytoma
            - output
                - urinary
                    - thiazides
            - features
                - levels <2.8 mmol/L
                    - polyuria and polydipsia
                    - dyspepsia
                        - due to calcium regulated release of gastrin
                    - depression
                    - mild cognitive impairment
                - levels <3.5 mmol/L
                    - all of the above plus:
                    - muscle weakness
                    - constipation
                    - anorexia and nausea
                    - fatigue
                - levels >3.5 mmol/L
                    - all of the above plus:
                    - abdominal pain
                    - vomiting
                    - dehydration
                    - lethargy
                    - cardiac arrhythmias, shortened QT interval
                    - coma
                    - pancreatitis
            - management
                - 0.9% sodium chloride to increase plasma volume and help to increase urinary output of calcium
                - loop diuretics such as furosemide may be considered
                - IV bisphosphonates after rehydration (e.g. pamidronate, zolendronic acid)
                - glucocorticoids for hypercalcaemia secondary to vitamin D toxicity, sarcoidosis or lymphoma
                - other specialist management options
    - hypocalcaemia
        - intake
            - calcium

        - vitamin D
        - phenytoin (increased metabolism of vitamin D)
    - redistribution
        - alkalosis
        - citrate toxicity
        - hyperphosphataemia
        - pancreatitis
        - tumour lysis syndrome
        - rhabdomyolysis
        - decreased bone turnover
        - hypoparathyroidism
        - drugs
            - bisphosphonates
            - PPIs
            - SSRIs
            - gentamicin
    - output
        - urinary
            - ethylene glycol
            - cisplatin
            - protamine
            - loop diuretics
        - non-urinary
            - bleeding
            - plasmapheresis
            - citrate renal replacement therapy
    - features
        - paraesthesia (usually fingers, toes and around mouth)
        - tetany
        - carpopedal spasm (wrist flexion and fingers drawn together)
        - muscle cramps
        - signs:
            - Chvostek's sign
                - muscle spasm provoked by tapping the facial nerve as it passes in front of the ear, below the zygomatic arch)
            - Trousseau's sign
                - inflate blood pressure cuff to above patient's systolic and maintain pressure for several minutes
                    - carpopedal spasm with flexion at the wrist and MCP joints, extension of the interphalangeal joints and adduction of the fingers
                    - they may have paraesthesia of the fingers and fasciculations
            - seizures
            - prolonged QT which may progress to VF or heart block

        - laryngospasm
        - bronchospasm
    - with prolonged hypocalcaemia:
        - subcapsular cataract
        - papilloedema
        - abnormal teeth
        - ectopic calcification (e.g. in basal ganglia, causing extrapyramidal symptoms)
        - dementia and confusion
- management
    - where symptomatic (seizures, tetany) or high risk of complications with a serum calcium <1.90 mmol/L:
        - 10 ml 10% calcium gluconate by slow IV injection
            - can be repeated or followed up with an infusion of 40ml/24 hours
    - oral supplementation with vitamin D
    - correct magnesium if needed first

- phosphate
    - hyperphosphataemia
        - intake
            - iatrogenic
            - vitamin D and A
        - redistribution
            - none
        - output
            - urinary
                - impaired renal phosphate excretion
            - non-urinary
                - rhabdomyolysis
                - hyperthermia
        - management
            - treat the underlying cause
            - restrict calcium phosphate intake
            - IV 0.9% sodium chloride if renal function normal
            - consider acetazolamide or a phosphate binder
            - may require dialysis if refractory
    - hypophosphataemia
        - intake
            - malnutrition
            - phosphate binders
            - vitamin D
            - malabsorption
            - TPN
        - redistribution
            - refeeding syndrome
            - insulin in DKA
        - output

- urinary
    - diuretics
    - osmotic diuresis
    - hyperparathyroidism
    - proximal tubular dysfunction (Fanconi syndrome)
- non-urinary
    - diarrhoea
    - sweat
    - burns
    - sepsis
    - bleeding

▪ features

- generally asymptomatic
- muscle weakness
- bone pain
- rhabdomyolysis
- altered mental status
- seizures
- focal neurological deficits
- congestive heart failure
- muscle pain

▪ management

- depends on cause and severity
    - mild cases are likely to resolve without treatment
    - severe cases can cause diaphragmatic weakness requiring ventilation
- oral supplements generally well tolerated
    - patients with eating disorders, dialysis, post-parathyroidectomy, alcohol excess should potentially be given prophylactic treatment
- no consensus of level at which IV treatment should be started
    - levels <0.3mmol/L should be treated
    - requires monitoring of calcium, phosphate and electrolyte levels every 6 hours as responses are unpredictable

- sodium
    - hypernatraemia

▪ causes

- pure free water loss (dehydration)
    - inadequate water intake
    - diabetes insipidus (cranial or neurogenic)
    - thirst impairment
        - dementia
        - hypothalamic lesions
- hypotonic fluid loss (dehydration + hypovolaemia)
    - dermal losses
        - burns
        - excessive sweating

        - gastrointestinal losses
            - non-secretory diarrhoea, laxative abuse
            - vomiting
            - NG drains
            - fistulas
        - urinary losses
            - loop diuretics
            - osmotic diuresis – e.g. hyperglycaemia
            - acute tubular necrosis – polyuric early stage
    - hypertonic (sodium gain – may cause hypovolaemia)
        - iatrogenic
            - use of hypertonic saline
            - tube feeding
            - IV antibiotics containing sodium
            - IV sodium bicarbonate
            - hypertonic dialysis
            - use of isotonic saline to replace losses in osmotic diuresis
        - excess salt ingestion
            - inadvertent – e.g. infant formula error
            - poisoning – more common in children (NAI)
        - hyperaldosteronism
    - intracellular shift of water (rare)
        - very strenuous exercise or electro-shock induced seizure causing a transient rise in cell osmolality so water moves into the cells
- features
    - in diabetes insipidus – thirst, polydipsia, polyuria
    - CNS dysfunction
        - lethargy
        - weakness
        - confusion
        - irritability
        - myoclonic jerks
        - seizures
    - symptoms and signs of dehydration and hypovolaemia
        - dry mouth
        - abnormal skin turgor
        - oliguria
        - tachycardia
        - orthostatic hypotension
- management
    - depends on severity and how acute the rise has been – assess clinical condition
    - address underlying cause where possible
        - stop GI fluid losses
        - control pyrexia

        - correct hyperglycaemia
        - withhold lactulose and diuretics
    - fluid replacement
        - oral or enteral where possible, with IV as last resort
        - water deficit
            - amount of water required to return serum sodium to normal
            - water deficit (L) = TBW x ((serum sodium/145) – 1)
            - TBW (total body water) = lean body weight (kg) x 0.6 for children and adult men, 0.5 for adult women and elderly men, 0.45 for elderly women
        - rate of replacement
            - when known to be chronic (>24h) or of unknown duration, correction should be no faster than 0.5mmol/L/hour or 10-12mmol/L/day
            - with rapid onset of hypernatraemia the aim should be to return to normal values within 24 hours
        - isotonic saline (0.9% sodium chloride) is appropriate to restore circulating volume if there is significant hypovolaemia
        - if hypervolaemic, diuretics and 5% dextrose can be given
        - hypotonic fluids should otherwise be used (0.45% sodium, 5% dextrose, oral water)
- hyponatraemia
    - different to others – mainly relates to distribution of water
        - need to consider:
            - osmolality
            - volume assessment
            - where the water is being lost from
    - hypotonic
        - hypovolaemic
            - urinary (high urinary sodium)
                - diuretics
                - osmotic diuretics
                - renal tubular acidosis
                - salt wasting
                - mineralocorticoid deficiencies
            - non-urinary (low urinary sodium)
                - upper GI – vomiting
                - mid GI – pancreatitis, bowel obstruction
                - lower GI – diarrhoea, bowel preparation
                - other – skin, bleeding, sweat
        - euvolaemic
            - SIADH (most common)
            - psychogenic polydipsia
            - hypotonic IV fluids
            - adrenal insufficiency
            - hypothyroidism

- hypervolaemic
    - congestive heart failure
    - cirrhosis
    - nephrotic syndrome
    - hypothyroidism
    - pregnancy
    - TURP syndrome
- isotonic (pseudohyponatraemia)
    - high proteins
    - high lipids
- hypertonic
    - glucose
    - mannitol
    - sorbitol
    - radiocontrast
    - advanced renal disease
- features
    - mild
        - anorexia
        - headache
        - nausea
        - vomiting
        - lethargy
    - moderate
        - personality change
        - muscle cramps and weakness
        - confusion
        - ataxia
    - severe
        - drowsiness
    - brainstem herniation can occur in acute severe hyponatraemia
        - coma
        - fixed unilateral dilated pupil
        - decorticate or decerebrate posturing
        - respiratory arrest
- management
    - stop any contributing medications/treat underlying causes
    - acute symptomatic hyponatraemia with CNS disturbance
        - level 2 monitored environment
        - hypertonic saline (3%)
            - 150ml IV over 15 minutes
            - repeat after 20 minutes if no clinical improvement
        - recheck serum sodium at 6, 12, 24 and 48 hours for overcorrection
            - no more than 10mmol/L in 24 hours
    - euvolaemic hyponatraemia

        - confirm – e.g. plasma osmolality <275 mOsm/kg, urine osmolality >100 mOsm/kg
        - urine sodium >20 – likely SIADH
            - investigate underlying cause (consider CT CAP + head)
            - calculate electrolyte free water clearance
                - (urine sodium+ potassium)/serum sodium
                - <0.5 – start 1 litre fluid restriction
                - 0.5-1.0 – start 0.5 litre fluid restriction
                - >1.0 – fluid restriction not advised, consult with endocrinologist
    - hypovolaemic hyponatraemia
        - treat with 0.9% sodium chloride
    - hypervolaemic hyponatraemia (oedema, raised JVP, LVF, ascites)
        - treat underlying cause

NepC4 fluid balance disorders

- osmolality
    - an estimation of the osmolar concentration of plasma
    - proportional to the number of particles per kilo of solvent
    - expressed as mOsmol/kg
    - normal osmolality of extracellular fluid is 280-295 mOsmol/kg
- osmolarity
    - an estimation of the osmolar concentration of plasma
    - proportional to the number of particles per litre of solution
    - expressed as mmol/L
    - derived from measured sodium, potassium, urea and glucose concentrations
    - unreliable in various conditions such as pseudohyponatraemia, hyperproteinaemia
    - calculations:
        - 2(Na+) + 2(K+) + glucose + urea (all in mmol/L)
        - 2(Na+) + glucose + urea (all in mmol/L)
- osmotic gap
    - an arbitrary measure of the difference between the actual osmolality (measured by the laboratory) and the calculated osmolality
    - usually less than 10-15 mOsmol/L
    - when increased, it indicates the presence of other osmotically active solutes which are not taken into account in calculated osmolality (e.g. methanol, ethylene glycol)
- clinical relevance
    - cell membranes in general are freely permeable to water
        - the osmolality of extracellular fluid is approximately equal to that of intracellular fluid
    - this means that changes in osmolality can cause problems with normal cell functioning and volume and even induce cytolysis
    - normally, increased blood osmolality stimulates secretion of ADH
        - results in increased water reabsorption, more concentrated urine and less concentrated blood plasma

        - diabetes insipidus is caused by hyposecretion of or insensitivity to the effects of ADH
    - low serum osmolality suppresses the release of ADH, resulting in decreased water reabsorption and more concentrated plasma
    - an increase of 2-3% in plasma osmolality produces a strong desire to drink
        - a change of 10-15% in blood volume and arterial pressure is required to produce the same response
- causes
    - normal or increased serum osmolality, increased urine osmolality
        - dehydration
        - renal disease and uraemia
        - congestive heart failure
        - Addison's disease
        - hypercalcaemia
        - diabetes mellitus/hyperglycaemia
        - hypernatraemia
        - alcohol ingestion
        - mannitol
    - normal or increased serum osmolality, decreased urine osmolality
        - diabetes insipidus
    - decreased serum osmolality, increased urine osmolality
        - syndrome of inappropriate ADH secretion (SIADH)
    - decreased serum osmolality, decreased urine osmolality (with no increase in fluid intake)
        - overhydration
        - hyponatraemia
        - adrenocortical insufficiency
        - sodium loss (diuretic or a low salt diet)
- diabetes insipidus
    - background
        - two major forms
            - cranial DI
                - decreased secretion of ADH
            - nephrogenic DI
                - decreased ability to concentrate urine because of resistance to ADH in the kidney
        - two other forms
            - gestational DI
                - results from degradation of vasopressin by placental vasopressinase
            - primary polydipsia (dipsogenic DI)
                - caused by a primary defect in the osmoregulation of thirst
                - has been reported in tuberculous meningitis, multiple sclerosis, neurosarcoidosis
    - aetiology
        - cranial DI
            - usually due to disease of the hypothalamus or surrounding tissues

- acquired
    - idiopathic
    - tumours
        - craniopharyngioma
        - germinoma
        - hypothalamic metastases (especially breast carcinoma)
        - hypothalamic glioma
        - large pituitary tumours with suprasellar extension
        - lymphoma
    - intracranial surgery
    - head injury
    - granulomata
        - sarcoidosis
        - TB
        - granulomatosis with polyangiitis
        - histiocytosis
    - infections
        - encephalitis
        - meningitis
        - cerebral abscess
    - vascular disorders
        - haemorrhage/thrombosis
        - aneurysms
        - sickle cell disease
        - Sheehan's syndrome (postpartum pituitary necrosis)
    - post-radiotherapy
- inherited
    - Wolfram's syndrome
        - autosomal recessive combination of DI, diabetes mellitus, optic atrophy, deafness
    - autosomal dominant mutations of vasopressin gene

- nephrogenic DI
    - acquired
        - idiopathic
        - hypokalaemia
        - hypercalcaemia
        - CKD
        - other metabolic derangements
        - drugs: e.g. ofloxacin, orlistat, lithium
        - renal tubular acidosis
        - pregnancy
            - renal hyposensitivity to ADH, increased placental elimination of ADH, lowered thirst threshold and effect of fluid retention
        - post-obstructive uropathy
    - congenital/genetic
        - X-linked mutation in V2 ADH receptor gene

        - autosomal recessive defect in aquaporin 2 gene
        - sporadic nephrogenic DI with general learning disability and intracerebral calcification
- symptoms
    - may be vague and insidious
    - marked polyuria
        - (>3 litres/day)
    - polydipsia and chronic thirst
        - may be a predilection for very cold drinks, usually water
    - nocturia several times per night
        - children may develop nocturnal enuresis where they had previously been dry
    - in infants: failure to thrive, irritability, protracted crying, fever, anorexia, fatiguability, feeding problems
    - urinary incontinence if there is damage to the bladder through chronic overdistension
- investigations
    - biochemistry
    - simultaneous plasma and urine osmolality
    - urine specific gravity
    - 24 hour urine collection
    - fluid deprivation test with response to desmopressin
    - MRI head or renal tract ultrasound may be considered
- management
    - cranial DI
        - physiological replacement with desmopressin
            - can be oral, intranasal or parenteral
        - mild cases can be managed by ingestion of water to quench thirst
        - chronic desmopressin overdose will cause hyponatraemia
    - nephrogenic DI
        - definitive treatment not always necessary
        - patients should have access to enough drinking water to quench their thirst
        - correction of metabolic abnormalities and stopping any contributing drugs
        - high dose DDAVP may be helpful in mild to moderate cases
        - combination therapy with a thiazide diuretic and NSAID may help to reduce volume of urine produced
- complications
    - DDAVP can worsen myocardial ischaemia in susceptible patients
        - may need nitrates or other anti-anginal medications
    - bladder dysfunction and hydroureter/hydronephrosis can occur if the condition is untreated for a period of time
- prognosis
    - usually good once treatment started
    - specialist follow up is required
    - death is rare in adults

        - patients with inborn errors are more likely to experience complications as the underlying cause cannot be removed
- SIADH
    - background
        - hyponatraemia due to an increase in concentration of ADH inappropriate to the current osmotic or volume status
    - diagnostic criteria
        - hypotonic hyponatraemia
        - urine osmolality > plasma osmolality
            - concentrated urine despite hypotonic blood
        - urinary sodium >20 mmol/L
        - normal renal, hepatic, cardiac, pituitary, adrenal and thyroid function
        - euvolaemia (absence of hypotension, hypovolaemia and oedema)
        - correction by water restriction
    - causes (MAD CHOP)
        - **m**ajor surgery
            - abdominal
            - thoracic
            - transsphenoidal pituitary surgery (6-7 days post op)
        - **A**DH production by tumours (ectopic)
            - small cell lung cancer
            - adenocarcinoma of pancreas/duodenum
            - leukaemia
            - lymphoma
            - thymoma
        - **d**rugs
            - antidepressants (e.g. SSRIs, TCAs, MAOIs)
            - psychotropics (e.g. haloperidol), carbamazepine, sodium valproate
            - anaesthetic drugs (barbiturates, inhalational agents), oxytocin, opioids
            - ADH analogues (vasopressin, DDAVP)
            - chemotherapy (vinca alkaloids, methotrexate, cyclophosphamide)
            - others (NSAIDs, amiodarone, ciprofloxacin, morphine, MDMA, PPIs)
        - **C**NS disorders
            - cerebral trauma
            - brain tumour (primary or metastases)
            - meningitis/encephalitis
            - brain abscess
            - SAH
            - acute intermittent porphyria
            - SLE
        - **h**ormone deficiency
            - hypothyroidism
            - adrenal insufficiency
        - **o**thers
            - Guillain-Barré syndrome
            - HIV infection

            - hereditary SIADH
            - giant cell arteritis
            - idiopathic
        - **p**ulmonary disorders
            - pneumonia (viral, fungal, bacterial)
            - TB
            - lung abscess
    - management
        - manage hyponatraemia
            - with incremental increase to avoid central pontine myelinolysis
        - fluid restriction
        - medications to decrease ADH secretion
            - e.g. demeclocycline

NepC5 renal replacement therapy

- indications for dialysis in AKI
    - clinical features of uraemia
        - e.g. pericarditis, gastritis, encephalopathy
    - hyperthermia
    - fluid retention leading to pulmonary oedema
        - inability to reduce excess volume with diuretics, with urine volume <200ml in 12 hours
    - severe hyperkalaemia (>6.5 mmol/L) unresponsive to medical management
    - serum sodium >155 mmol/L or <120 mmol/L
    - severe acid base disturbance (pH <7.0) not controlled by sodium bicarbonate
    - severe renal failure (urea >30 mmol/L, creatinine >500 μmol/L)
    - toxicity with drugs that can be dialysed
        - e.g. methanol, isopropanol, ethylene glycol, lithium, salicylate, theophylline, valproic acid
- renal replacement options
    - haemofiltration
        - blood is pumped through an extracorporeal system incorporating a semi-permeable membrane
        - the hydrostatic pressure created on the blood side of the filter drives plasma water across the filter
            - this is ultrafiltration
        - molecules small enough to pass through the membrane (<50,000 Daltons) are dragged across the membrane with the water by process of convection
        - the filtered fluid (ultrafiltrate) is discarded and a replacement fluid added in an adjustable fashion according to the desired fluid balance
        - venovenous RRT requires a double-lumen vascular catheter placed in a central vein with the tip in the IVC for femoral lines or SVC (1-2cm from the right atrium) for internal jugular and subclavian lines
            - internal jugular is the straightest route (especially right side) and overall preferred site but may be occupied by other lines
            - subclavian is the cleanest site and most comfortable for the patient; can lead to pneumothorax and subclavian vein stenosis

            - femoral is fairly straight and can provide good flows but has the highest risk of infection, particularly in obese patients
        - complications include:
            - complications related to the vascath (including line related sepsis)
            - haemodynamic instability
            - air emboli
            - platelet consumption
            - blood loss
            - electrolyte imbalances
            - hypothermia
            - effects of anticoagulation
- haemodialysis
    - blood is pumped through an extracorporeal system that incorporates a dialyser
    - in the dialyser, blood is separated from a crystalloid solution (dialysate) by a semi-permeable membrane
    - solutes move across the membrane along their concentration gradient from one compartment to the other according to Fick's law of diffusion (e.g. bicarbonate moves from dialysate to blood whereas urea and potassium move from blood to dialysate)
    - to maintain concentration gradients and enhance the efficiency of the system, dialysate moves countercurrent to the flow of blood
    - when removal of water is required, the pressure on the blood side of the membrane has to be increased to force water molecules to pass into the dialysate
    - requires an arteriovenous fistula (ideally fashioned 3-6 months before starting dialysis) or a permanent vascath (internal jugular or subclavian)
    - can be daily or three times a week
    - complications include:
        - access-related
            - local infection, endocarditis, osteomyelitis, stenosis, thrombosis, aneurysm
            - hypotension, cardiac arrhythmia, air embolism
            - nausea and vomiting, headache, cramps
            - fever – infected lines
            - dialyser reactions – anaphylaxis to sterilising agents
            - heparin induced thrombocytopaenia, haemolysis
            - disequilibration syndrome – restlessness, headache, tremors, fits, coma
            - depression
- peritoneal dialysis
    - an option for CKD
    - dialysate is infused into the peritoneal cavity
        - the blood flowing through peritoneal capillaries acts as the blood source
    - ultrafiltration is controlled by altering the osmolality of the dialysate solution to draw water out of the patient's blood

- achieved with glucose or other large molecular weight solutes in the dialysate
    - glucose load can cause poor diabetes control and weight gain
- a permanent catheter is inserted into the peritoneum under local or general anaesthetic
- waste solutes are removed by exchanging the peritoneal fluid for a fresh solution
- patients can be trained to perform continuous ambulatory peritoneal dialysis
    - usually involves four exchanges of around 20 minutes spaced throughout the day
    - alternatively, automated peritoneal dialysis can be used to do a number of exchanges overnight while the patient is asleep, then only one or two daytime exchanges are required
- peritoneal dialysis can be performed at home, at work or whilst on holiday, however a lot of support is still required
- contra-indications:
    - intra-abdominal adhesions and abdominal wall stoma
    - obesity, intestinal disease, respiratory disease and hernias are relative contra-indications
- complications:
    - peritonitis, sclerosing peritonitis
    - catheter problems: infection, blockage, kinking, leaks or slow drainage
    - constipation, fluid retention, hyperglycaemia, weight gain
    - hernias (incisional, inguinal, umbilical)
    - back pain
    - malnutrition
    - depression

- transplantation
    - provides the best long term outcome for patients with end stage kidney disease
        - 85-90% are from cadaveric donors, the rest from living donors
    - age should not prevent consideration of transplant but comorbidities adversely affect survival
    - the transplanted kidney is normally placed extraperitoneally in the iliac fossa, with the native kidney left in place
    - patients require initial immunological T cell suppression, then long term immunosuppression to prevent rejection
    - they are followed up for life with annual screening for cancers, drug toxicity and cardiovascular disease
    - contra-indications for transplantation
        - cancer
        - active infection
        - uncontrolled ischaemic heart disease
        - acquired immunodeficiency disease with opportunistic infections

        - acute viral hepatitis
        - extensive peripheral vascular disease
        - mental incapacity
    - benefits of transplantation
        - can stop dialysis
        - improved quality of life with normal diet and activity, relaxation of fluid restriction
        - reversal of anaemia and renal bone disease
    - risks of transplantation
        - immediate operative complications (local infection, pain, pneumonia, deep vein thrombosis
        - immediate graft failure
        - arterial or venous thrombosis in the transplant
        - infections (viral, bacterial, fungal)
        - cancer (skin, lymphoma)
        - side-effects of immunosuppressive drugs
    - complications of transplantation and subsequent immunosuppression treatment
        - postoperative problems
            - e.g. DVT, PE, pneumonia
        - opportunistic infections
            - viral
                - particularly herpes simplex in first four weeks and CMV later
            - fungal
            - bacterial
        - malignancies
            - especially lymphoma, skin cancer
        - drug toxicity, bone marrow suppression
        - recurrence of the original disease in the transplant
        - urinary tract obstruction
        - cardiovascular disease, hypertension, dyslipidaemia
        - graft rejection
            - hyperacute
                - occurs within minutes of insertion
                - now rare due to more accurate cross matching
                - requires removal of graft
            - accelerated
                - aggressive mainly T-cell mediated crisis occurring within a few days in patients previously sensitised
                - presents with fever, swollen transplanted kidney and rapidly increasing serum creatinine
                - can be salvaged with high-dose steroids and antilymphocyte antibodies but long-term survival is affected
            - acute cellular

        - occurs in around 25% of patients usually in 1-3 weeks
            - can occur up to 12 weeks
        - clinical signs are fluid retention, rising blood pressure and rapid increase in creatinine
        - treatment is with IV steroids after diagnosis by biopsy
    - chronic
        - presents with gradual rise in serum creatinine and proteinuria, resistant hypertension
        - graft biopsy shows vascular changes, fibrosis and tubular atrophy
        - not responsive to increasing immunosuppression therapy
- prognosis
    - survival of transplant recipient at ten years for cadaveric and live donor transplants is 71% and 89% respectively
    - acute rejection and early graft loss are becoming less common
    - risk of graft failure, return to dialysis and death increases with:
        - cadaveric donor renal transplant
        - more human leukocyte antigen (HLA) mismatches
        - increased donor age
        - cold ischaemia time greater than 24 hours
        - history of diabetic nephropathy

NEUROLOGY

NeuP1 acute confusion

- definition
    - acute, generalised brain dysfunction (cerebral insufficiency)
    - ICD-10 definition:
        - an aetiologically non-specific organic cerebral syndrome characterised by concurrent disturbances of consciousness and attention, perception, thinking, memory, psychomotor behaviour, emotion and the sleep-wake schedule; duration is variable and the degree of severity ranges from mild to very severe
    - key features:
        - acute (not dementia)
        - causes inattention (e.g. disorientation, inability to perform complex tasks)
        - tends to wax and wane and may have lucid periods
        - diagnosis is clinical
    - occurs in 30% of ED patients
    - incidence is higher in those with pre-existing cognitive impairment
- common features
    - classification
        - hyperactive delirium

                - agitation
            - hypoactive delirium
                - patient is withdrawn, mute, drowsy
                - may go unnoticed as not overtly problematic
            - mixed
                - periods of hyperactive and hypoactive delirium
    - features can involve:
        - psychotic symptoms
            - hallucinations, delusions, paranoia
        - emotional symptoms
            - fear, anxiety, irritability, anger
        - day-night reversal with hyperactivity at night (sundowning)
        - increased sympathetic activity
            - hypertension, tachycardia
- risk factors
    - age ≥65
    - male sex
    - pre-existing cognitive deficit
    - severity of dementia
    - severe comorbidity
    - previous episode of delirium
    - operative factors
        - e.g. hip fracture repairs, emergency operations
    - certain conditions:
        - burns
        - AIDS
        - fractures
        - infection
        - low albumin
        - dehydration
    - current hip fracture or severe illness
    - drug use and dependence
    - substance misuse
    - extremes of sensory experience (e.g. hypothermia, hyperthermia)
    - visual or hearing problems
    - poor mobility
    - social isolation
    - stress
    - terminal illness
    - movement to a new environment
    - ICU admission
    - urea/creatinine abnormalities
- common causes
    - CVA
        - haemorrhage
        - ischaemic stroke
    - infection

        - any systemic illness in elderly patients
        - meningitis, encephalitis
        - sepsis
    - medications
        - entire list should be reviewed
            - particularly new medications, medications acting on the nervous system, possible drug interactions
        - examples include:
            - GABAergics
                - e.g. benzodiazepines, muscle relaxants
            - antihistamines
                - e.g. diphenhydramine, promethazine
            - opioids
            - antimicrobials
                - e.g. fluoroquinolones
            - anticonvulsants
                - e.g. carbamazepine, phenytoin, valproate
            - Parkinson's medications
            - metoclopramide
            - sleep medications
            - steroids
    - metabolic
        - hypoglycaemia
        - fever/hyperthermia
        - electrolyte abnormality
            - especially abnormal sodium or hypercalcaemia
        - Wernicke's encephalopathy
        - hyperammonemia not due to cirrhosis
    - organ failure
        - cardiovascular
            - shock
            - hypertensive encephalopathy
        - pulmonary
            - hypoxaemia
            - hypercapnia
        - liver
            - hepatic encephalopathy
        - renal
            - uraemia
        - endocrine
            - thyroid storm
            - myxoedema coma
        - haematological
            - thrombotic thrombocytopenic purpura
    - seizure
    - toxicological
        - withdrawal, e.g.:

                - alcohol
                - benzodiazepines
                - gabapentin
                - baclofen
                - opioids
                - serotonin-noradrenaline receptor inhibitors
            - intoxication/poisoning e.g.:
                - carbon monoxide
                - lithium
                - digoxin
        - sleep deprivation
            - noisy environment
            - frequent disturbance for observations
            - uncontrolled pain
- assessment
    - Confusion Assessment Method (CAM)
        - acute onset and fluctuating course AND
        - inattention (e.g. 20-1 test with reduced capacity to shift attention or keep attention focused) AND EITHER
        - disorganised thinking (disorganised or incoherent speech) OR
        - changed level of consciousness (e.g. lethargic or in state of stupor
    - history
        - full history
            - including collateral and cognition testing
        - examination
            - including looking for sources of infection including ENT and lymphadenopathy and checking for constipation
        - investigations
            - bloods
                - basics
                    - fingerprick glucose
                    - electrolytes (including calcium, magnesium, phosphate)
                    - FBC
                - other possible tests depending on presentation
                    - LFTs
                    - ammonia
                    - TSH
                    - ABG/VBG if hypercapnia suspected
                    - urinalysis/blood cultures if suspicion of infection
                    - pertinent drug levels
                        - e.g. digoxin, lithium, theophylline
                    - additional toxicology
            - neuroimaging
                - non-contrast CT
                    - consider for:
                        - delirium with unclear history/precipitant

                - CNS trauma
                - significant anticoagulation
                - focal neurological signs
                - substantially reduced GCS
            - MRI
        - lumbar puncture
            - may be useful for patients presenting with delirium
        - EEG may be considered in some circumstances
- management
    - basic management
        - avoid admission if possible and the patient has sufficient community support
    - supportive measures
        - clear communication
        - reminders of day, time, location and identification of people
        - clock in view
        - familiar objects from home
        - glasses, walking aids, hearing aids
        - consistency of staff where possible
        - relaxation
        - involvement of family and carers
    - environmental measures
        - avoid sensory extremes (over or under stimulation)
        - adequate space and sleep
        - single room if possible
        - avoidance of jargon
        - reduction of excess noise
        - control of lighting including low light at night
        - control of room temperature
        - interpreters where needed
        - maintain walking in ambulatory patients
        - adequate nutrition and attendance to continence
        - management of wandering
            - consider reasons (e.g. need for toilet)
            - rectify causes or use distraction
            - aim to keep patient safe using least restrictive measures
            - avoid restraint or sedation wherever possible – may worsen the situation
            - try to involve relatives/carers
    - medical management
        - drugs can cause adverse effects and worsen delirium
        - antipsychotics may be considered in certain patients, particularly aggressive patients not responding to de-escalation techniques
        - haloperidol or olanzapine are preferred
            - lowest possible dose
                - titrated gradually until symptoms resolve
            - both are unlicensed uses in the UK

                - both can cause extra-pyramidal effects and should be avoided in at-risk patients such as those with Parkinson's disease or Lewy-Body dementia
                - shortest possible time (normally one week or less)
        - post-discharge
            - symptoms may last longer than the underlying condition
                - some patients will be discharged with underlying abnormalities, e.g. disorientation, inattention, depression
            - families and carers may also require support
- complications
    - hospital-acquired infections
    - pressure sores
    - fractures (e.g. from falls)
    - residual psychiatric and cognitive impairment
- prognosis
    - research suggests that short-term delirium has no effect on mortality
        - delirium lasting >30 days has a two to three fold increase in mortality
    - some patients do not recover for months
    - a third continue to have delirium and may require institutional care
- prevention
    - NICE recommendations include:
        - multidisciplinary approach to prevention
        - assessment of patients within 24 hours of admission noting factors that may precipitate or worsen delirium
        - interventions including:
            - appropriate lighting and regular orientation
            - promotion of cognitively stimulating activities
            - regular visits from people known to the patient
            - identification and correction of hypoxia
            - pain
                - verbal and non-verbal assessment
                - management
            - daily medication review and stopping non-essential medication
            - other factors including:
                - dehydration
                - constipation
                - reduced mobility
                - infection
                - poor nutrition
                - sensory impairment
                - sleep disturbance

NeuP2 headache

- primary headache
    - background
        - primary headache is the cause of 90% of headaches presenting to the ED

        - primary headaches are those where the specific aetiology is not fully understood (e.g. migraine)
            - secondary headaches have a clear and understandable origin (e.g. ruptured aneurysm)
    - history
        - further investigation is required if any of the following are present:
            - sudden onset – 'thunderclap' headache
            - 'worst headache ever'
            - new headache in the elderly
            - loss of consciousness
            - headache associated with activity
    - examination
        - needs to include:
            - cognitive state
            - vital signs
            - neck movement
            - pupil symmetry
            - fundi
            - motor function including pronator drift
            - gait
        - further investigation required if any are abnormal
    - red flags:
        - significantly increase the risk that there is a secondary cause
        - warrant further investigation
        - include:
            - headache in someone >50 years
            - thunderclap headache
                - reaching maximum intensity within 60 seconds of onset
            - headaches increasing in severity or frequency
            - headache with fever, neck stiffness or reduced level of consciousness
            - focal neurological symptoms or signs
            - papilloedema
            - headache after trauma
    - tension headaches
        - most common type of primary headache
        - around 3% of people are chronic sufferers
        - pain is usually band-like across the forehead
            - can radiate from the neck, back or eyes
        - typically last 4-6 hours
        - often have identifiable triggers
            - e.g. stress, hunger, sleep deprivation, eye strain
        - there is risk of medication overuse headaches or rebound headache if patients frequently use analgesia
        - management
            - ascertain that the patient is not:
                - over using analgesic medication

        - showing evidence of drug dependency
        - showing signs of depression
    - episodic tension-type headaches respond well to over the counter analgesics, with ibuprofen more effective than paracetamol
    - prophylactic options for chronic tension-type headaches include amitriptyline and sodium valproate
    - other management includes:
        - regular exercise
        - stretching
        - balanced meals
        - adequate sleep
- migraine
  - background
    - usually begins under age 40
    - more common in women
    - classically unilateral (seen in 60%) and with aura (seen in 20%)
        - commonest auras are visual (e.g. rings, flashes, blurring)
        - paraesthesia is also a relatively common aura
  - pathophysiology
    - undetermined
    - it is now thought that changes in blood flow are probably the result rather than the cause of the migraine
    - the most probable hypothesis is that it is a disorder of brain function rather than blood vessels
  - diagnosis
    - there are 5 screening criteria:
        - pulsating
        - duration 4-72 hours
        - unilateral
        - nausea
        - disabling
    - likelihood ratio for migraine is 3.5 if three features are present and 24 if four are present
  - any change in pattern of migraine in known sufferers requires thorough re-evaluation
  - management
    - analgesia
        - aspirin, NSAIDs and paracetamol are effective
    - anti-emetics
        - metoclopramide and domperidone relieve nausea and gastric status and enhance the bio-availability of oral medications
    - non-specific therapies
        - chlorpromazine (25-50mg IM) and prochlorperazine (10mg IV or IM) have been successfully used as single agent therapies
    - specific therapies

        - triptans
            - most effective if taken when the headache is mild
            - ineffective if taken before the onset of symptoms
        - patients with migraine not responding to triptans should be considered for treatment with ergotamine tartrate 1-2mg
- cluster headaches
    - much more common in men (6:1)
    - onset is usually in the mid 20s
    - typical symptoms include:
        - sharp, stabbing unilateral pain around the orbit or temporal area
        - lacrimation
    - onset is acute with rapid escalation of pain
    - pain may last from a few minutes to several hours
    - each cluster lasts a few weeks
    - most sufferers have two to three clusters per year
    - management:
        - high flow oxygen therapy is usually effective
            - 10 litres/min for 15 minutes
        - triptans (e.g. sumatriptan 6mg SC) can also be used
- exertional headaches
    - associated with physical activity
        - particularly strenuous activity such as:
            - weightlifting
            - running
            - sexual intercourse
    - typically become severe quite quickly
    - may also become severe as a result of:
        - coughing
        - sneezing
        - straining with bowel movements
    - most exertional headaches are benign
        - some are related to pathologies such as SAH or brain tumour
    - patients presenting with recent onset exertional headache require urgent CT +/- MRI to exclude structural brain lesions
    - management:
        - exclude structural lesion at first presentation
        - advise patient of benign nature
        - some patients benefit from beta-blockers
- trigeminal neuralgia
    - most common between 50 and 70 years
    - most cases are idiopathic
    - causes brief electric shock-like pains in the distribution of one or more branches of the trigeminal nerve
    - episodes are often triggered by minimal stimuli
    - there are no neurological signs on examination
    - management:
        - drugs shown to be effective include:

        - carbamazepine
        - phenytoin
        - valproate
        - lamotrigine
        - gabapentin
      - around 30% do not respond to drug therapy and may require surgical intervention
  - investigation
    - not-required for patients with normal neurological examination, non-thunderclap headache and no red flags
      - CT scan for the 10% of ED patients where history and/or examination suggests a secondary cause
- secondary headaches
  - red flags and possible causes
    - headache in person >50 years
      - mass lesion
      - temporal arteritis
    - thunderclap headache
      - SAH
      - pituitary apoplexy
      - haemorrhage into mass lesion or vascular malformation
      - posterior fossa mass lesion
    - headaches increasing in severity and frequency
      - mass lesion
      - subdural haematoma
      - medication overuse
    - headache with fever, neck stiffness or reduced level of consciousness
      - meningitis
      - encephalitis
      - cerebral vasculitis
    - focal neurological signs or symptoms
      - mass lesion
      - vascular malformation
      - stroke
    - papilloedema
      - mass lesion
      - benign intracranial hypertension
    - headache after trauma
      - intra-cranial haemorrhage
        - subarachnoid
        - extradural
        - subdural
      - concussion
  - subarachnoid haemorrhage
    - background
      - commonest cause of non-traumatic presentation in under 20s is arteriovenous malformation

        - rupture of cerebral artery aneurysm is the commonest cause in older patients
- risk factors
    - female gender
    - hypertension
    - positive family history
    - polycystic kidney disease
- history
    - sudden severe thunderclap headache
    - worst ever headache
    - associated with nausea, vomiting, photophobia, neck stiffness (70%)
    - retinal haemorrhage may be seen in severe cases
    - neurological symptoms:
        - seizures
        - diplopia
        - brief loss of consciousness
        - reduced level of consciousness
        - focal neurological deficit (20%)
            - commonest deficit is a cranial nerve III palsy
- Ottawa rule out criteria for SAH
    - SAH is ruled out if there is not:
        - age ≥40 years
        - neck pain or stiffness
        - witnessed loss of consciousness
        - onset during exertion
        - thunderclap headache (instantly peaking pain)
        - limited neck flexion on examination
- grading (Hunt and Hess scale)
    - 1 – asymptomatic, or minimal headache and slight nuchal rigidity
    - 2 – moderate to severe headache, nuchal rigidity and no neurological deficit except cranial nerve palsy
    - 3 – drowsy, minimal neurological deficit
    - 4 – drowsy, significant neurological deficit
    - 5 – deep coma, decerebrate rigidity and moribund appearance
- investigation
    - CT 98% sensitive within 12 hours

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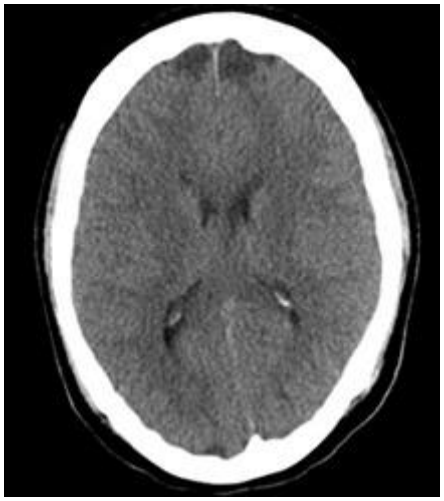
        - blood localised to the basal cisterns or in the Sylvanian or interhemispheric fissures suggests rupture of an aneurysm
        - blood overlying the cerebral convexities or within the superficial brain parenchyma suggests rupture of an AV malformation
      - 1 in 50 patients scanned within 12 hours have a false negative result so LP is required to look for xanthochromia
  - prognosis
    - around 50% die from the haemorrhage or as a consequence of secondary complications
    - 50% of those who survive are severely disabled
    - outcome is directly related to neurological status at presentation
- cerebral venous sinus thrombosis
  - background
    - uncommon condition, seen more commonly in women
    - relative dehydration is a common feature
    - the cavernous sinus is most commonly affected
      - usually as a complication of infection of the middle third of the face
  - causes
    - infective
      - TB
      - sepsis
      - endocarditis
    - non-infective
      - dural puncture
      - neurosurgery
      - pregnancy and contraceptive use
      - severe dehydration
      - head injury
  - history

- symptoms are often subacute
- headache is usually diffuse, progressive and persistent and often present for over 48 hours
- in 15% it is described as a thunderclap headache
- seizures and focal deficits often occur

- clinical examination
  - focal neurological signs
  - papilloedema
  - reduced visual acuity
  - chemosis (conjunctival oedema)
  - proptosis
  - ophthalmoplegia (cranial nerves III to VI)
- investigation
  - plain CT may appear normal
    - may show:
      - small ventricles
      - hyper-density of the thrombosed sinuses
      - small haemorrhages secondary to venous congestion



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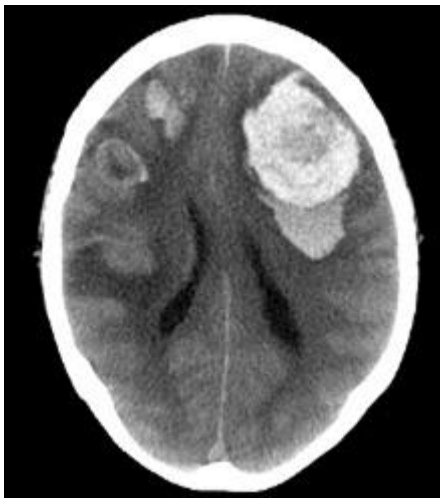
  - CT or MRI venogram is the investigation of choice
  - if the diagnosis is suspected, MRI is required
- management
  - treatment of underlying cause
  - heparin to reduce risk of clot extension
    - increases risk of cerebral haemorrhage
  - in patients with deteriorating conscious level, a neuro-radiologically guided catheter for thrombolytic therapy should be considered
  - phenytoin for patients presenting with seizures
- prognosis
  - not directly related to neurological status at presentation

- mortality is approximately 15%
    - a similar number survive with persistent deficits

- brain tumours
    - background
        - most are metastatic from extra-cranial primaries
            - the commonest are:
                - lung
                - breast
                - kidney
                - melanoma
        - headache normally occurs by one of three mechanisms:
            - meningeal involvement
            - vascular involvement
            - raised intracranial pressure
    - presentation
        - headache is a presenting feature in 50%
            - maximal on waking and easing through the course of the day
            - may wake the patient from sleep
            - exacerbated by straining or bending
        - seizures in 25%
            - more common in patients with gliomas
        - friends and relatives may describe behavioural changes and cognitive deficits
        - on examination there is weakness or focal deficit in two thirds of patients
    - investigation
        - MRI is the investigation of choice
        - CT with contrast is probably the best ED option



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        - radiological differentiation between tumours and abscesses is difficult and requires clinical correlation

        - management
            - 80% of primary brain tumours are high-grade gliomas (glioblastoma multiforme)
                - median survival time without treatment is 3 months
                - with optimal treatment less than 25% survive for 2 years
                - death occurs due to cerebral oedema or raised intracranial pressure
            - brain metastases occur in up to 30% of cancer patients
                - mean survival is <12 months
            - for primary and secondary lesions, ED management is usually limited to steroid therapy to reduce oedema and relieve mass effect symptoms
                - steroids are particularly useful where there is extensive vasogenic oedema
                - optimal dose of dexamethasone is 4mg/day
                    - improves symptoms and function as well as higher doses whilst minimising side effects
    - intra-cerebral abscess
        - 25% occur in children
            - either as a result of otogenic focus or cyanotic congenital heart disease
        - most common organism are Streptococci (especially Strep milleri); anaerobes are also isolated in 50% of cases
        - fungal infections are most commonly seen in immunocompromised patients
            - usually involve candida species
        - HIV patients have a high incidence of toxoplasmosis
        - in 20% the cause is unknown
        - patients can present with indolent or fulminant symptoms
            - fever may or may not be present
        - symptoms depend on the site and size of the abscess
            - altered mental state and hemiplegia are common
        - investigation
            - it is not possible to distinguish reliably between primary tumour, solitary metastasis and cerebral abscess on plain CT alone
            - CT with contrast shows:
                - a hypodense lesion
                - peripheral ring enhancement
                - further hypodense surrounding area of brain oedema

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- idiopathic intracranial hypertension
  - causes
    - unknown
    - associations with:
      - female gender
      - oral contraception
      - anabolic steroids
      - vitamin A excess
      - obesity
      - tetracyclines
  - presentation
    - daily headache
      - bifrontotemporal
      - worse on straining
    - transient visual loss may occur due to compression of the optic nerves
      - visual impairment is permanent in 10%
    - examination is often normal
      - papilloedema may be detected
      - there may be a visual field deficit
      - a CN VI nerve palsy is the most common focal sign
  - diagnosis (modified Dandy criteria)
    - signs and symptoms of raised ICP; CSF pressure >25cm H_2O
    - no localising signs except CN VI nerve palsy
    - normal CSF composition
    - normal to small (slit) ventricles on CT with no intracranial mass
  - management
    - withdrawing 5-10ml of CSF usually relieves pressure and eases the headache
    - the ultimate goal is to reduce CSF pressure to <200mmHg or until symptoms resolve

- if there is evidence of visual impairment, management needs to be more aggressive and includes:
    - urgent referral to ophthalmology
    - treatment with furosemide and acetazolamide to reduce CSF production
    - possible surgery

- carbon monoxide poisoning
    - patients presenting to the ED with headache should be asked about exposure to carbon monoxide, e.g. from faulty vehicle exhausts or poorly maintained/inadequately ventilated gas appliances
    - particularly likely if more than one person from the same household presents with headache
    - other symptoms include:
        - somnolence
        - nausea
        - cherry red appearance (late and peri-mortem sign)
    - diagnosis is with measurement of carboxyhaemoglobin levels on ABG
    - management
        - 100% oxygen through a close fitting mask
        - hyperbaric oxygen therapy should be considered for patients with a reduced level of consciousness
- carotid and vertebral artery dissection
    - background
        - associated with trauma, which may be minor (e.g. chiropractic manipulation)
        - hypertension and Marfan's syndrome are known associations
        - one of two pathological events may occur:
            - haemorrhage into the vessel wall leading to occlusion of the vessel which may then cause cerebral oedema
            - an intimal tear; may cause ischaemia or result in embolic phenomena minutes to months after the initial event
                - TIAs are common
                - if the vertebral arteries are implicated, evidence of brainstem dysfunction will occur
    - presentation
        - abrupt onset of unilateral headache, neck or face pain
        - brainstem and cerebellar signs with vertebral artery dissection
        - Horner's syndrome
            - may be seen with vertebral and carotid artery dissections
            - diagnosis should be considered in any patient <50 years presenting with symptoms of stroke
            - presents with small pupil and drooping eyelid
    - investigation
        - plain CT head is often normal
        - CT or MRI angiography is required
- pituitary apoplexy
    - causes

- associated with haemorrhage into an enlarged pituitary gland
- most commonly in patients with pituitary adenoma
- most common in middle aged patients and men
- Sheehan's syndrome:
    - rare complication of pregnancy
    - pituitary gland undergoes necrosis following significant peri- or post-partum haemorrhage
    - most women present subacutely with features of pituitary insufficiency (e.g. difficulties with lactation, features of hypothyroidism)
    - some present acutely with electrolyte disturbances such as hyponatraemia due to disordered ADH production

▪ presentation
- abrupt severe headache (95%)
- progressive visual loss
- later, there may be signs of pituitary insufficiency
- compression of the cavernous sinus causes compression of the II, IV and VI cranial nerves as they cross the sinus, causing diplopia

▪ diagnosis
- requires high index of suspicion
- should be considered with headache and ophthalmoplegia
- haemorrhage should be detectable on CT

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o temporal arteritis

▪ background
- peak incidence is in patients over 70
- rare in patients under 50
- if untreated, visual loss occurs in one third
- there is an association with polymyalgia rheumatica

▪ presentation
- over 80% present with headache

        - may be continuous or intermittent
        - typically worse after cold exposure
        - localised to the frontotemporal region
        - sharp or throbbing
    - associated symptoms include:
        - jaw claudication
        - fatigue
        - fever
        - weight loss
        - night sweats
        - diplopia
        - amaurosis fugax
    - classically, there is tenderness to palpation over the temples or loss of pulsation of the temporal artery
- investigations
    - should include ESR and referral for temporal artery biopsy
- treatment
    - high dose steroids should be started immediately without waiting for results of investigations
        - prednisolone 1mg/kg/day
        - steroids do not interfere with biopsy findings if the biopsy is undertaken within a week of starting medication

- pre-eclampsia
    - 25% of cases occur post-partum
    - treatment is with antihypertensives (labetolol, nifedipine) plus magnesium sulphate if seizures occur (4g over 5-15 minutes followed by an infusion of 1g/hour)

NeuP3 seizures/status epilepticus

seizure types (international classification of epileptic seizures)

- focal seizures
    - originate within networks limited to one hemisphere
    - discretely localised or more widely distributed
    - divided into simple focal (motor or sensory) with retained awareness, and focal dyscognitive seizures (impaired awareness)
    - simple focal seizures (no loss of consciousness)
    - focal dyscognitive seizures
        - with impairment of consciousness at onset
        - simple focal onset followed by impairment of consciousness
    - focal seizure evolving to generalised tonic-clonic convulsions
- generalised seizures
    - convulsive or non-convulsive with bilateral discharges involving subcortical structures
    - associated with impairment of consciousness and distortion of electrical activity of the whole or a large part of both sides of the brain
    - may be tonic-clonic, isolated tonic or clonic, myoclonic or absence
    - absence

    - myoclonic
    - clonic
    - tonic
    - tonic-clonic
    - atonic
- unclassified epileptic seizures (usually used when an adequate description is not available)

symptoms of seizures

    - sudden falls
    - involuntary jerky movements of the limbs whilst awake
    - blank spells
    - unexplained incontinence of urine with loss of awareness or in sleep
    - odd events occurring in sleep (e.g. fall from bed, jerky movements, automatisms)
    - episodes of confused behaviour with impaired awareness
    - possible simple focal seizures
    - epigastric fullness sensation
    - déjà vu
    - premonition
    - fear
    - elation, depression
    - depersonalisation, derealisation
    - inability to understand or express language (written or spoken)
    - loss of memory, disorientation
    - olfactory, gustatory, visual, auditory hallucinations
    - focal motor or somatosensory deficit, or positive symptoms (jerking, tingling)

first seizure

    - background
        - treatment is not normally recommended until after a second seizure but may be indicated after the first seizure if the patient has a neurological deficit, brain imaging shows a structural abnormality, EEG shows unequivocal epileptic activity or the individual or their family considers the risk of further seizure unacceptable
        - detailed history should exclude previous myoclonic, absence or focal seizures
    - epidemiology
        - there is an 8-10% lifetime risk of one seizure and a 3% chance of epilepsy
        - 50% of patients with apparent first seizure have had other minor seizures
    - risk factors
        - 25-30% of first seizures have an underlying cause; provoking factors include:
            - fever
            - head injury
            - excessive alcohol intake
            - withdrawal from alcohol or drugs
            - hypoglycaemia
            - electrolyte disturbance
            - brain infection (meningitis, encephalitis)

            - ischaemic stroke
            - intracranial haemorrhage
            - eclampsia
            - proconvulsive drugs (e.g. tramadol, theophylline, baclofen)
        - seizures may be triggered by specific stimuli in susceptible individuals with an underlying epilepsy disorder
            - e.g. stroboscopic lights, reading, severe psychological stress, sleep deprivation
- presentation
    - detailed history from the patient and any witnesses is essential
    - videos and written descriptions can be helpful
    - tongue biting and postictal confusion may suggest a seizure
    - full assessment should include cardiac, neurological and mental state assessment
- differential
    - syncope (post anoxic convulsion)
    - TIA
    - metabolic encephalopathy (including hypoglycaemia or electrolyte disturbance)
    - sleep-walking
    - night terrors
    - complex migraines
    - cardiac arrythmias
    - psychogenic non-epileptic seizures
- investigation
    - bloods (in adults)
        - glucose
        - electrolytes
        - calcium
        - U&Es
        - LFTs
    - 12 lead ECG
    - neuroimaging
        - MRI is the investigation of choice but CT can be used to identify gross pathology
    - EEG
        - shows substantial abnormalities in 70% of cases if performed within 24-48 hours of a first seizure
        - sleep-deprived EEG can detect epileptiform discharges in a further 13-31%
        - usually performed after the second seizure
- management
    - specialist assessment (e.g. first fit clinic) as soon as possible
    - advice for family and carers on how to recognise and manage seizures
    - the patient should be advised to stop driving whilst waiting to see the specialist and to avoid potentially dangerous work or leisure activities (avoid swimming, ensure bathing is undertaken with supervision)

        - continued restrictions should be assessed on an individual basis
            - working with dangerous machines should probably be suspended for at least 6 months
        - driving is permitted after one year's freedom from seizures after an unprovoked seizure and on a case by case basis for provoked seizures
        - commercial driving after an unprovoked seizure is usually not permitted until 10 years' freedom from seizure with anti-epileptic drug treatment
    - anti-epileptic drug treatment
        - should only be started once the diagnosis of epilepsy is confirmed, except in exceptional circumstances
            - overall risk of recurrence after one seizure is 30-40%, greatest in the first 12 months and falls to <10% after two years
            - early treatment does not appear to alter the prognosis of epilepsy
            - prognosis is best predicted by number of seizures within the first 6 months after diagnosis and response to first anti-epileptic drug
        - should be started by a specialist
        - generally recommended after a second seizure
    - driving
        - all patients should be advised to stop driving and inform the DVLA and their motor insurance company after a seizure
        - Group 1 (private car or motorcycle)
            - 6 months off driving from the date of the seizure unless there are clinical factors or investigation results which suggest an unacceptably high risk of further seizure
        - Group 2 (large goods vehicle or passenger carrying vehicle)
            - five years off driving from the date of the seizure if the licence holder has undergone recent assessment by a neurologist and there are no clinical factors or investigation results (e.g. EEG, brain scan) which indicate that the risk of further seizure is >2% per annum
            - they should have taken no anti epileptic medication during the five year period
    - prognosis
        - risk factors for seizure recurrence include:
            - static or progressive brain abnormalities
            - focal neurological findings
            - focal or generalised epileptiform activity on EEG
            - status epilepticus
            - family history of epilepsy
            - previous febrile seizures
        - a first seizure caused by an acute disturbance of brain function is unlikely to recur (3-10%)
        - if unprovoked, 30-50% will recur
            - 60-70% of recurrences are within 6 months of the first seizure
        - after a second unprovoked seizure, 70-80% will recur, justifying the diagnosis of epilepsy
        - seizures associated with reversible metabolic or toxic disturbances are associated with a minor risk of subsequent epilepsy

- seizures provoked by disorders that cause permanent damage to the brain, such as abscess, have a higher risk of recurrence (10%)

status epilepticus

- background
  - mortality approaches 30% if left untreated
  - presentations vary from clinically obvious tonic-clonic convulsions to subtle focal seizures to sensory alterations associated with partial seizures
  - traditional definitions are either:
    - a single seizure lasting for more than 30 minutes
    - multiple seizure of shorter duration without full neurological recovery between seizures
  - there is evidence that seizures persisting for 5 minutes are likely to go on for more than 30 minutes
    - the average seizure duration in adults is <1 minute so 5 minutes is a deviation from the norm
    - there is a suggestion that neuronal injury can occur after 5 minutes of seizure activity
  - the term impending status epilepticus is sometimes used for seizures lasting more than 5 minutes or intermittent seizures without full recovery
    - this recognises the need for immediate treatment but acknowledges that not all are established status epilepticus and up to 40% may terminate spontaneously within 30 minutes
  - established status epilepticus
    - clinical or electrographic seizures that persist for 30 minutes or longer without full neurological recovery in between
  - refractory status epilepticus
    - persistence of convulsions despite adequate doses of two intravenous agents
- pathophysiology
  - seizures result from rapid abnormal electrical discharges from cerebral neurones
  - thought to arise from an imbalance between excitatory and inhibitory neurotransmitters (commonly glutamate and GABA) leading to failure of the inhibitory process
  - numerous systemic and metabolic changes occur in association with prolonged seizures:
    - tachycardia
    - hypertension
    - hyperglycaemia
    - lactic acidosis
  - most are thought to result from a catecholamine surge and will resolve with seizure resolution
  - beyond 30 minutes cerebral autoregulation may become impaired and cerebral perfusion fall as hypotension occurs with potential for ischaemic injury and cerebral oedema
- aetiology

        - chronic epilepsy
            - one third have a background of chronic epilepsy
            - status usually results from:
                - drug non-compliance
                - drug withdrawal
                - drug therapy alteration
            - may also occur in the presence of:
                - intercurrent illness
                - metabolic abnormalities
                - co-ingestion of drugs that lower the seizure threshold
        - future epilepsy onset
            - in one third of patients the episode represents the onset of future epilepsy
        - no prior or future history
            - in one third of patients, the episode occurs with no prior or future history of epilepsy
            - may result from:
                - drug withdrawal syndromes (alcohol, barbiturates, benzodiazepines)
                - acute structural brain injury (e.g. stroke, subarachnoid haemorrhage, trauma, cerebral hypoxia)
                - CNS infection (meningitis, encephalitis, abscess)
    - classification
        - generalised tonic-clonic status epilepticus
            - most clinically obvious presentation
            - sudden powerful contraction of the muscles (tonic phase), often associated with a fall to the floor, followed by regular rhythmic contraction and relaxation (clonic phase) of the musculature of all limbs
            - invariably associated with alterations in consciousness
        - partial status epilepticus
            - seizures originating from a specific area of the cortex without any impairment of consciousness
            - presentation may be far more subtle than generalised seizures
            - may involve motor (regular rhythmic contractions of a single limb or muscle group) or sensory (illusions, hallucinations, depersonalisation) symptoms
            - complex partial seizures are associated with a degree of conscious impairment and arise from a single region of the brain
                - symptoms may include aphasia, apraxia and focal limb weakness
                - may be associated with verbal or motor automatisms
        - other forms of status epilepticus exist:
            - absence
            - myoclonus
            - pseudo-status epilepticus
    - investigation

        - bloods
            - U&Es
            - magnesium
            - calcium
            - FBC
        - VBG to assess metabolic status
        - other investigations depending on circumstances:
            - CT head
            - blood cultures
            - serum anticonvulsant levels
            - lumbar puncture
        - EEG
            - rarely of use in the acute setting
            - may be useful in suspected non-convulsive status or in patients who are paralysed and sedated
    - management
        - principles:
            - early initiation of anticonvulsants is key to successful treatment
            - outcomes worsen with increasing duration of status epilepticus
            - loss of responsiveness to benzodiazepines will occur with time
        - pre-hospital
            - airway management
            - high flow oxygen where available
            - blood glucose with IV dextrose if required
            - benzodiazepines
                - rectal diazepam or buccal/intranasal midazolam
        - in hospital
            - benzodiazepines
                - first line of agents
                - prevent propagation of seizure rather than affecting the original focus
                - all have rapid onset of action due to lipid solubility and the consequent ability to cross the blood-brain barrier
                - most frequently used agents:
                    - diazepam
                        - lipid soluble
                        - available as IV and rectal preparations
                        - rectal diazepam terminates seizures in 70% of patients
                        - IV diazepam will terminate seizures in 60-80%
                    - lorazepam
                        - less lipophilic
                        - smaller volume of distribution and longer intracerebral half life (12 hours) than diazepam

                - may therefore provide more prolonged suppression of seizures
                - IV lorazepam terminates seizures in 60-90% of patients
            - midazolam
                - water soluble so can be used as buccal or intranasal preparations
                - primarily used in the pre-hospital setting
        - all benzodiazepines carry a risk of respiratory depression and hypotension
- hydantoins
    - phenytoin
        - long-acting drug – prevents seizure recurrence over an extended period of time
        - delayed onset (10-30 minutes) – requires use alongside a rapidly acting agent such as a benzodiazepine
        - not water soluble
        - formulated with propylene glycol which can cause significant side effects including:
            - local infusion site reactions
            - arrhythmias mandating cardiac monitoring during infusion
            - hypotension
            - purple glove syndrome
                - local oedema, skin discolouration and pain distal to the infusion site
            - skin necrosis and limb ischaemia have been reported
        - complications are reduced by keeping infusion rates below 50mg/minute and by avoiding co-infusion with dextrose containing fluids which can lead to the formation of precipitates
    - fosphenytoin
        - the inactive prodrug of phenytoin
        - broken down into the active drug by serum phosphatases
        - water soluble so associated with fewer side effects
        - can be infused at a faster rate
            - however time taken to achieve therapeutic phenytoin levels is similar due to the time taken to form the active metabolite
- paraldehyde
    - recommended in many treatment algorithms as a second line agent after benzodiazepines in the absence of IV access
    - given rectally mixed with an equal volume of olive oil

        - evidence for it is lacking but there is a large body of anecdotal evidence
    - barbiturates
        - similar mode of action to benzodiazepines by modification of the actions of GABA
        - not standard first line drugs; may be used for refractory status epilepticus
        - agents include:
            - phenobarbital
            - thiopental
            - pentobarbital
                - short-acting agent with a rapid onset of action that is very effective in terminating seizures
    - other agents
        - IV Pabrinex or thiamine
            - for patients with known or suspected alcohol abuse or poor nutritional status
        - propofol
            - rapid onset and short duration
            - effective at terminating seizures
            - use is limited in children due to 'propofol infusion syndrome'
            - hypotension can result from high doses
        - levetiracetam
            - not metabolised in the liver
            - causes less cardiovascular and respiratory depression than other agents
        - midazolam
            - can rapidly control seizures when used as a continuous infusion
- step by step procedure
    - on arrival
        - check ABC
        - high flow oxygen
        - check blood glucose
        - IV access: lorazepam 0.1mg/kg IV
        - no IV access: diazepam 0.5mg/kg PR
    - at 10 minutes
        - IV access: lorazepam 0.1mg/kg IV
        - no IV access: paraldehyde 0.4ml/kg (in same volume of olive oil) PR
    - at 20 minutes
        - request senior help if not already present
        - consider IO access or venous cutdown if no IV access

        - phenytoin 18mg/kg IV OR phenobarbitone 20mg/kg IV AND paraldehyde 0.4ml/kg (in same volume of olive oil) PR if not already given
    - at 40 minutes
        - rapid sequence intubation
        - transfer to ITU
        - thiopental 4mg/kg

sudden unexplained death in epilepsy (SUDEP)

- sudden, unexpected, unwitnessed, non-traumatic, non-drowning death of a person with epilepsy, with or without a seizure, excluding documented status epilepticus and in whom post-mortem examination does not reveal a structural or toxicological cause of death
- incidence is between 0.09 and 0.35/1000 patient years
- generalised tonic-clonic seizures are the principal risk factor for SUDEP
    - early identification and management of treatment resistant epilepsy may reduce incidence
- SUDEP is the most common cause of death directly related to epilepsy and most frequently occurs in people with chronic epilepsy
- patients with epilepsy and their carers should be provided with information about the small but definite risk of SUDEP
- SUDEP seems to occur more commonly during sleep
    - more often affects young adults with intractable epilepsy, those with neurological comorbidity and patients receiving anti-epileptic drug polytherapy
- risk can be minimised by optimising seizure control and being aware of the potential consequences of nocturnal seizures

NeuP4 speech disturbance

- background
    - dysarthria is a disorder of speech, dysphasia is a disorder of language
    - speech is the process of articulation and pronunciation
        - it involves the bulbar muscles and the physical ability to form words
    - language is the process in which thoughts and ideas become spoken
        - it involves the selection of words to be spoken (semantics) and the formulation of appropriate sentences or phases (syntax)
- definitions
    - dysarthria
        - a speech disorder caused by disturbance of muscular control
    - dysphasia
        - impairment of language, may coexist with dysarthria
        - can be receptive (difficulty in comprehension) or expressive (difficulty in putting words together to make meaning)
    - a person with pure dysarthria without dysphasia would be able to read and write as normal and make meaningful gestures
- dominant hemisphere
    - the speech area is in the left side of the brain in 99% of right-handed people

        - the remaining 1% may be left-handers who have been forced to use their right hand
    - in left-handed people the right hemisphere is dominant in 30%
    - impairment of the speech area in a stroke causing left-sided weakness is rare and occurs in virtually no right-handers and only 30% of left-handers
    - as a general rule, a lesion of the left hemisphere causes dysphasia whilst a lesion of the right hemisphere causes neglect, visuo-spatial and cognitive problems
- epidemiology
    - aetiology is damage or disease of the brain so it is most common with advancing age
    - disease is usually vascular, neoplastic or degenerative
    - 85% of cases arise from strokes and around a third of people who have strokes have dysphasia
    - in younger people it is usually the result of a head injury
- dysarthria
    - causes
        - upper motor neurone lesions of the cerebral hemispheres or lower motor neurone lesions of the brain stem
        - also results from disruption to the integrated action of upper motor neurones, basal ganglia and cerebellum
    - features
        - slurred and weak articulation with a weak voice is typical of pseudobulbar palsy from a stroke
            - other neurological signs are usually unilateral with a right-sided hemiplegia
            - brain stem stroke may lead to bilateral signs with dysarthria or anarthria
        - slurred, scanning and staccato speech caused by cerebellar lesions is typical of multiple sclerosis
        - a dysrhythmic, dysphonic and monotonous voice is caused by disease of the extrapyramidal system in Parkinson's disease
            - movement is rigid and stiff and that includes phonation
        - indistinct articulation, hypernasality and bilateral weakness caused by lower motor neurone disorders can occur with motor neurone disease
    - management
        - speech and language therapy to assess and treat the bulbar and facial muscles
        - patients may also require advice on alternative methods of communication
- dysphasia
    - causes
        - due to a lesion of the dominant hemisphere
        - may include impaired ability to read, write and use gestures
        - most common cause is cerebrovascular disease, but can arise from space occupying lesions, head injury or dementia
    - features
        - it is a disruption in the links between thought and language
        - diagnosis is only made after excluding sensory impairment of vision or hearing, perceptual impairment (agnosia), cognitive impairment (memory),

impaired movement (apraxia) or thought disturbance (e.g. dementia, schizophrenia)
        - the patient's ability to repeat difficult phrases or tongue twisters can be indicative
        - people with receptive dysphasia often have language that is fluent with a normal rhythm and articulation but it is meaningless as they fail to comprehend what they are saying
        - people with expressive dysphasia are not fluent and have difficulty forming words and sentences
            - there are grammatical errors and difficulty finding the right word
            - in severe cases they do not speak spontaneously but they usually understand what is said to them
        - specific types are associated with damage to particular cortical regions but in practice distinctions are not always clear
            - generally, expressive dysphasia suggests an anterior lesion and receptive dysphasia suggests a posterior lesion
    - specific types
        - sensory (Wernicke's) dysphasia/aphasia
            - lesions are in the left posterior perisylvian region
            - primary symptoms are general comprehension deficits, word retrieval deficits and sematic paraphrasias
            - lesions damage the semantic content of language whilst leaving the language production function intact
            - the consequence is fluent speech lacking in content
            - patients lack awareness of their speech difficulties
        - production (Broca's) dysphasia/aphasia
            - lesions are located in the left pre-central areas
            - non-fluent or expressive dysphasia
            - there are deficits in speech production, prosody and syntactic comprehension
            - patients typically have slow and halting speech with good semantic content
            - comprehension is usually good
            - patients are aware of their language difficulties
        - conduction dysphasia/aphasia
            - lesions are around the arcuate fasciculus, posterior parietal and temporal regions
            - symptoms are naming deficits, inability to repeat non-meaningful words and word strings with apparently normal speech comprehension and production
            - patients are aware of their difficulties
        - deep dysphasia/aphasia
            - lesions are in the temporal lobe, especially those mediating phonological processing
            - symptoms are word repetition problems and semantic paraphrasia (semantically related word substituted when asked to repeat a target word)

        - transcortical sensory dysphasia/aphasia
            - lesions are located between Broca's area and the supplementary motor area
            - symptoms are transient mutism, telegrammatic speech (omitting unimportant words) and dysprosodic (monotone) speech
        - global dysphasia/aphasia
            - occurs with extensive damage to the left perisylvian region, white matter, basal ganglia and thalamus
            - symptoms are extensive with generalised deficits in comprehension, repetition, naming and speech production
    - examination
        - tests for receptive dysphasia may include asking patients to read words or passages
            - they are then asked to explain the words or passage
        - comprehension of spoken material is assessed by asking the patient to listen to a passage and explain it or to follow instructions such as 'point to the door'
        - tests for expressive dysphasia include:
            - asking the patient to name a series of objects and some of their parts (e.g. 'what is this?', 'what part of the watch is this?')
            - if language is limited, you can hold up objects and ask 'is this a pen?' for different objects, therefore requiring different answers
                - look for difficulty in finding the correct word and perseveration
            - can the patient talk spontaneously on a familiar topic (e.g. family, work)?
            - can the patient count numbers or recite the days of the week, write a brief dictated passage, write a brief spontaneous passage, copy a short passage?
                - bearing in mind comorbid status
    - management
        - referral to speech and language therapy
            - assessment of the nature of the problem and tailored exercises to encourage the recovery of fluent speech and understanding
    - prognosis
        - 40-60% of stroke survivors may retain some degree of dysphasia
            - it is associated with lower independence, less social participation, poorer rehabilitation outcomes, worsened quality of life
        - severe dysphasias are unlikely to show much improvement but other forms can show rapid improvement
        - probability of recovery following trauma is higher than following stroke

NeuP5 hemiparesis/hemiplegia

- hemiparesis
    - examination
        - inspection
        - gait and Rhomberg's

        - tone
        - power
        - reflexes
        - sensation
        - co-ordination
        - cranial nerves
        - carotid bruits
        - functional assessment – can the patient do up a button/hold a pen?
    - possible findings include:
        - increased tone
        - pronator drift
        - pyramidal weakness
        - increased reflexes
        - clonus
        - upgoing plantar
        - hemiparetic gait
        - upper limb may be in flexion with lower limb extended
        - sensory loss
        - UMN facial weakness (forehead sparing)
    - differential includes:
        - stroke
        - space occupying lesion
            - tumour
            - subdural haemorrhage
            - abscess
        - hemiplegic cerebral palsy (present from birth)
        - Todd's paresis post-seizure
        - hemiplegic migraine
        - stroke mimic
            - sepsis
            - hypoglycaemia
            - demyelination
    - possible investigations:
        - bloods
            - FBC
            - U&E
            - LFTs
            - CRP/ESR
            - lipids
            - fasting glucose/HbA1c
            - coagulation
        - ECG
        - CXR
        - urine dipstick for blood and protein
        - CT head
            - early signs of ischaemic stroke include:
                - loss of grey-white differentiation

        - insular ribbon sign
        - sulcal asymmetry
        - hyperdense MCA sign
    - carotid dopplers if anterior circulation stroke suspected
    - echo
    - may require MRI +/- MRA
    - later investigations may include vasculitis and thrombophilia screen, HIV and syphilis serology
- Todd's paresis
    - follows a seizure
    - characterised by a temporary, usually unilateral, weakness of hand, arm or leg after partial seizure activity affecting that limb
        - Todd's paralysis is a more severe, less common form
        - may also affect speech or vision
        - usually resolves within 48 hours
        - cause is unknown
- hemiplegic migraine
    - rare condition
    - attacks of headache and motor weakness
        - weakness is usually unilateral but may be bilateral and can switch sides
        - may be associated with impaired consciousness, cerebellar ataxia and intellectual disability
        - aura symptoms such as sensory or visual symptoms are common
    - motor symptoms usually last <72 hours
    - differential is broad
        - includes:
            - cerebrovascular disease
            - cerebral amyloid angiopathy
            - epilepsy with hemiparesis
            - brain tumour
            - post-radiation therapy stroke-like migraine
            - transient headache with neurological deficits and CSF lymphocytosis
            - alternating hemiplegia of childhood
            - CNS infection
            - hereditary and metabolic disorders
        - neuroimaging, CSF analysis and EEG are used along with genetic testing
    - management relies on control of triggering factors and sometimes hospitalisation
        - abortive treatments may be helpful when started early but evidence is lacking

NeuP6 gait abnormality

- hemiplegic gait
    - description
        - unliteral weakness on the affected side
        - arm flexed, adducted and internally rotated
        - leg on same side is in extension with plantar flexion of the foot and toes

        - the patient holds their arm to one side and drags the affected leg in a semicircle (circumduction) due to weakness of distal muscles (foot drop) and extensor hypertonia in lower limb
        - in mild hemiparesis, loss of normal arm swing and slight circumduction may be the only abnormalities
    - causes
        - most commonly seen in stroke
- diplegic (spastic) gait
    - description
        - involvement on both sides with spasticity in the lower extremities worse than the upper extremities
        - characteristic extreme tightness of hip adductors
        - legs can cross in the midline due to hip adductor tightness (scissoring gait)
            - patients may have hip adductor release surgery
        - abnormally narrow base, dragging both legs and scraping the toes
    - causes
        - bilateral periventricular lesions such as cerebral palsy
- neuropathic gait (steppage/equine gait)
    - description
        - seen in patients with foot drop (weakness of foot dorsiflexion)
        - cause is due to an attempt to lift the leg high enough so the foot does not drag on the floor
    - causes
        - unilateral
            - peroneal nerve palsy
            - L5 radiculopathy
        - bilateral
            - amyotrophic lateral sclerosis
            - Charcot-Marie-Tooth disease
            - other peripheral neuropathies (including from uncontrolled diabetes)
- myopathic gait (waddling)
    - description
        - hip girdle muscles are responsible for keeping the pelvis level whilst walking
        - weakness on one side leads to a drop in the pelvis on the contralateral side while walking (Trendelenburg sign)
        - with bilateral weakness there is dropping of the pelvis on both sides leading to waddling
    - causes
        - myopathies, e.g. muscular dystrophy
- choreiform gait (hyperkinetic)
    - description
        - irregular, jerky, involuntary movements in all extremities
        - walking may accentuate the baseline movement disorder
    - causes
        - basal ganglia disorders including Sydenham's chorea, Huntington's disease, other forms of chorea, athetosis or dystonia

- ataxic gait (cerebellar)
    - description
        - clumsy, staggering movements with a wide based gait
        - the patient's body may swagger back and forth whilst standing still (titubation)
        - patients cannot walk heel to toe or in a straight line
        - patients with truncal instability are more likely to have midline cerebellar disease at the vermis
    - causes
        - cerebellar disease
        - acute alcohol intoxication
- Parkinsonian gait (festinating/propulsive gait)
    - description
        - the patient has rigidity and bradykinesia
        - they will be stooped with the head and neck forwards, with flexion at the knees
        - the whole upper extremity is in flexion, with the fingers usually extended
        - the patient walks with slow, little steps
        - they may have difficulty initiating the steps
        - they may have an involuntary inclination to take accelerating steps (festination)
    - causes
        - Parkinson's disease
        - any other condition causing parkinsonism such as side effects from drugs
- sensory gait
    - description
        - an ataxic gait occurring because of loss of proprioceptive input
        - the patient slams the foot hard on the ground to know when the feet land and their location
        - it is exacerbated when patients cannot see their feet (e.g. in the dark)
        - sometimes referred to as a stomping gait
        - can resemble cerebellar ataxia when severe
    - causes
        - disorders of the dorsal columns
            - B12 deficiency
            - tabes dorsalis (neurosyphilis)
        - diseases affecting the peripheral nerves, e.g. uncontrolled diabetes

NeuP7 visual disturbance

- identification of the presenting complaint
    - blurred vision
        - a single image seen indistinctly
        - identify if it is at distance, near or both
    - decrease in peripheral vision
        - the patient may describe bumping into things or frequent scrapes when parking the car
    - alteration of a clear image

        - e.g. microscopia/macroscopia (image appears smaller or bigger)
        - metamophosia (distorted image)
    - interference with a clear image
        - e.g. floaters, flashes of light (photopsia)
    - diplopia
        - monocular, binocular, horizontal, vertical, oblique
    - other disturbances of vision
        - iridescent vision (haloes, rainbows)
        - dark adaptation problems or night blindness (nyctalopia)
        - colour vision abnormalities
- history
    - unilateral or bilateral
    - sudden or gradual in onset
        - if sudden, what the patient was doing at the time
        - if gradual, over what period of time
    - whether it has happened before, and if so, when and what
        - whether it has been diagnosed
    - whether there are any associated factors
        - pain (ocular or head)
        - associated ocular complaints
            - e.g. red eye, discharge, abnormal appearances
        - systemic complaints
            - e.g. headache, other neurological problems, malaise
    - other important factors in the history:
        - other ocular history
            - whether it could be worsening of a pre-existing condition (e.g. cataracts becoming symptomatic) or a new condition arising from a recent problem (e.g. infection after cataract surgery)
        - medical history
        - medication
            - drugs may be toxic to the eye or precipitate acute angle closure glaucoma
        - family history
            - atopy
            - diabetes
            - thyroid disease
            - certain malignancies
            - any hereditary syndromes
        - social history
            - metal work
            - elderly people with cataracts making it difficult for them to cope alone
            - HGV drivers who need to contact the DVLA
- examination
    - assessment of structure
        - work from front to back, starting with the lids and examining as far back as possible

                - anterior segment, lens and vitreous through to the fundus
            - corneal sensation
            - fluorescein examination of cornea
            - red reflex
                - if absent, the problem is likely to be within the visual media (cornea, crystalline lens, vitreous)
                - if present, the problem is more likely to lie at the level of the retina or optic nerve
            - look for evidence of trauma, infection or inflammation
        - assessment of function
            - visual acuity
                - mandatory in both eyes
                - note strength of glasses if worn
                - establish whether acuity is improved by a pinhole
            - pupil examination
                - use a dimly lit room
                - patient should look at the far wall to overcome the accommodation reflex
                - a bright light source should be directed from below to avoid nose shadow
                - compare size and shape of pupils and assess for direct response to light
                - swinging flashlight test to look for afferent pupillary defect
            - confrontational visual field test
                - examines peripheral vision
            - external ocular movements
            - Amsler grid to examine the central vision and assess macular function
        - other examination
            - guided by history and ocular examination
            - may include:
                - full neurological examination
                - examination of pulse for atrial fibrillation
                - listening for carotid bruits
                - checking blood pressure
                - checking urine for glucose
- investigation
    - depends on what is suspected
    - may require same day ophthalmology review
    - may need blood tests such as ESR/plasma viscosity if temporal arteritis suspected
- differential
    - unilateral, sudden and painful
        - trauma
            - history
            - associated injuries
            - inflammation +/- hyphaema
        - orbital cellulitis
            - hot, red, swollen and tender area surrounding the eye

            - systemically unwell patient
        - endophthalmitis
            - associated with accidental or surgical trauma
            - may be recent, old or endogenous
            - painful red eye, reduced visual acuity, hypopyon
        - corneal problems
            - trauma
            - infection
            - severe dry eye or exposure keratopathy
            - contact lens problems
        - anterior uveitis
            - red eye associated with photophobia, headache
            - may have had previous episodes
        - acute angle closure glaucoma
            - often precipitated when pupil in mid-dilation (e.g. watching tv in dim light)
            - often associated with systemic malaise (headache, nausea, vomiting)
        - arteritic anterior optic neuropathy (temporal arteritis)
            - patients >50 years
            - pain is often more of a headache than acute eye pain
            - other features include jaw claudication, scalp tenderness, polymyalgia rheumatica, anorexia, weight loss, fever
        - optic neuritis
            - can be bilateral
            - painful presentation of multiple sclerosis
            - pain, particularly on moving the eye
            - may be other focal neurological symptoms
        - migraine
            - when there are scintillations, the pain in the head often appears when the visual disturbance is ebbing or has disappeared
    - unilateral, sudden and painless
        - ocular causes
            - vitreous haemorrhage
                - may present as sudden floaters
                - may arise as a result of diabetic retinopathy, a retinal break or detachment, retinal vein occlusion and occasionally a posterior vitreous detachment or age-related macular degeneration
                - should be considered in trauma, subarachnoid or subdural haemorrhage, intraocular tumours and sickle cell disease
            - central retinal artery occlusion
                - painless, almost instantaneous reduction of vision in one eye
                - degree of visual impairment is usually considerable and there may be complete loss
            - central retinal vein occlusion

        - frequently presents with loss of vision or blurred vision, often starting on waking
    - AMD
        - usually the dry form which is associated with a progressive decrease in visual acuity
        - in 10% of cases the wet form occurs, where a neovascular membrane forms, which may be susceptible to bleeding, causing dramatic and rapid loss of vision
    - retinal detachment
        - tends to produce a curtain across the visual field
    - intermediate or posterior uveitis
        - tends to present with marked floaters +/- blurring of vision
    - anterior ischaemic optic neuropathy
        - relative afferent pupillary defect, pale and oedematous optic disc, flame-shaped haemorrhages and possibly an altitudinal visual field defect
    - hydrops
        - acute corneal oedema may occur in a number of conditions such as keratoconus
    - cerebrovascular disease
        - usually causes visual disturbance in both eyes but should be considered with unilateral blurred vision
- systemic causes
    - giant cell arteritis
        - usually progressive but rapid unilateral loss of vision
    - papilloedema
        - may cause headache rather than eye pain
        - visual abnormalities tend to be transient initially
    - amaurosis fugax
        - characteristically presents as a curtain across the vision
        - may be associated with intraocular emboli, atrial fibrillation and carotid bruit
    - migraine prodrome
        - may occur without a following headache
        - usually unilateral but may progress to be homonymous
- bilateral, sudden and painful
    - arc eye
        - in welders
        - there will probably be a history of welding a number of hours earlier without adequate eye protection
    - malignant hypertension
        - progressive blurring of vision bilaterally (not necessarily equally) in a susceptible patient
        - pain tends to be in the form of headaches
- bilateral, sudden and painless
    - papilloedema
    - cerebrovascular disease

- may lead to damage to the visual pathways and optic cortex
- there may or may not be macular sparing
- visual disturbance is often homonymous

- drugs
  - anticholinergics
  - sedative drugs such as antipsychotics and anticonvulsants
- refractive errors
  - tend to change very slowly over years
    - may change more rapidly in poorly controlled diabetes

- unilateral or bilateral, gradual and painless
  - glaucoma
    - asymptomatic but progressive peripheral visual field loss, usually bilateral but asymmetric
  - refractive errors
    - can be affected by hormonal changes such as pregnancy but reverts on restoration of baseline hormone levels
    - progression of corneal disease such as dystrophies or keratoconus
  - cataracts
    - patient may complain of dulling of colours (or relatives noticing a predilection for gaudy colours)
    - risk factors include diabetes, systemic steroids, immunosuppression
  - AMD
    - dry form
  - cystoid macular oedema
    - as a result of surgery, inflammation or vascular disease
  - diabetic maculopathy
    - ischaemia may lead to gradual decrease in visual acuity whereas oedema tends to result in more acute visual distortion
  - genetic disease
    - may affect elements of the visual media (e.g. the cornea in keratoconus) or the retina (e.g. Best's disease)
  - drug toxicity
    - e.g. hydroxychloroquine, methanol, ethambutol, COX-2 inhibitors
  - other toxic agents
    - e.g. organophosphates
  - inflammatory optic neuropathies
    - tend to be associated with systemic diseases such as sarcoid, vasculitis, syphilis
  - chronic eye strain
    - e.g. from excessive use of computer screens under adverse conditions
    - may cause blurred vision
- unilateral, gradual and painful
  - neoplastic or inflammatory disease of the orbit and globe
- medically unexplained visual loss
  - can occur in four circumstances:
    - organic disease has not been diagnosed

- the patient seeks secondary gain
- visual loss is psychosomatic
- fabricated or induced illness

- management
  - depends on cause
  - generally, acute painful conditions require same day referral to ophthalmology
  - DVLA advice for driving should be checked
- visual field defects
  - causes
    - central field loss (degeneration of the fovea)
      - age-related macular degeneration
      - optic neuropathy
      - Leber's optic atrophy
      - macular holes
      - cone dystrophies
      - retinal artery occlusion
      - rare conditions such as Best's disease, Stargardt's disease and achromatopsia
    - peripheral field loss
      - glaucoma
      - retinal detachment
      - retinitis pigmentosa
      - chorioretinitis
      - branch retinal artery occlusion
  - types of visual field loss
    - central or peripheral
    - scotoma
      - a defect surrounded by a normal visual field
      - may begin as a gradual enlargement of the blind spot and go unnoticed until quite large
      - relative scotoma
        - an area where objects of low luminance cannot be seen but larger or brighter ones can
      - absolute scotoma
        - nothing can be seen at all within that area
    - hemianopia (binocular deficit in each eye's hemifield)
      - bitemporal hemianopia
        - the two halves lost are on the outside of each eye's peripheral vision
        - effectively creates a central visual tunnel
      - homonymous hemianopia
        - the two halves lost are on the corresponding area of the visual field in both eyes (i.e. left or right half of the visual field)
      - altitudinal hemianopia
        - the dividing line between loss and sight is horizontal with visual loss either above or below the line

                - quadrantanopia
                    - incomplete hemianopia referring to a quarter of the visual field
                - sectoral defect
                    - incomplete hemianopia
        - location of lesions
            - lesions at the level of the retina
                - affect one eye only
            - lesions before the chiasm
                - produce a field deficit in the ipsilateral eye
                - field defects from damage to the optic nerve tend to be central, asymmetrical and unilateral
                - visual acuity is often affected
            - lesions at the chiasm
                - classically produce a bitemporal hemianopia
            - lesions after the chiasm
                - produce homonymous field deficits
                - a lesion in the right optic tract produces a left visual field defect
                - fibres in the optic tract gradually rotate until the fibres reach the geniculate body, so lesions before the geniculate body may produce incongruous lesions
            - occipital lobe lesions
                - if both lobes are injured, the patient has cortical visual impairment
                    - the patient cannot process visual information and behaves in a similar fashion to someone who has developed a peripheral visual loss
                    - some patients try to behave as if they still have vision (Anton's syndrome)
                - lesions of the primary visual perception area of the right or left occipital lobe will produce a clear loss of visual perception from the contralateral visual field
                    - patients are usually aware of the deficit and do not neglect that side of the visual field
        - safety to drive
            - patients with a newly diagnosed visual field defect should not drive until it has been formally assessed
            - patients must contact the DVLA who will arrange a specific visual field test (Estermann's visual field test) carried out by an approved optometrist

NeuP8 weakness/paralysis

- background
    - acute non-traumatic weakness may occur as a result of a wide variety of underlying aetiologies, many of which are life-threatening
    - key considerations include:
        - assessment of the need for intubation and respiratory support
        - identification of time critical emergencies

        - determination of the underlying cause, based on clinical assessment and investigations, including careful physical examination to facilitate neurological localisation and diagnosis
- factors to consider in the decision to intubate
    - general
        - increasing generalised muscle weakness
        - dysphagia
        - dysphonia
        - dyspnoea on exertion and at rest
    - subjective
        - rapid shallow breathing
        - tachycardia
        - weak cough
        - interrupted speech (gasping for air)
        - use of accessory muscles
        - abdominal paradoxical breathing
        - orthopnoea
        - weakness of trapezius and neck muscles – inability to lift head from bed
        - inability to perform single breath count – count from 1 to 10 in single exhalation (roughly equivalent to FVC <1.0 L)
        - cough after swallowing
    - objective
        - decreased level of consciousness
        - hypoxaemia
        - vital capacity <1 L or 20ml/kg, or 50% decrease in VC in a day
        - maximum inspiratory pressure >-30 cmH2O
        - maximum expiratory pressure < 40 cmH2O
        - hypercapnia (occurs late)
    - time course and anticipated trajectory of illness should also be taken into account
        - intervention should ideally be before the onset of hypercapnia
- immediately life-threatening causes
    - stroke syndromes
    - aortic dissection
    - CNS infections
    - spinal cord syndromes
    - seizure disorders
    - envenoming (e.g. snake)
    - acute toxicity (e.g. organophosphates, heavy metals)
    - electrolyte/metabolic disorders
- differential diagnosis
    - overview
        - myopathic
            - rhabdomyolysis
            - ischaemic
            - congenital
            - CIW
        - neuromuscular junction

            - myasthenia gravis
            - botulism
        - neuropathic
            - GBS
            - CIW
            - polio
        - spinal cord
            - infarct
            - infection
            - tumour
        - brain stem
            - haemorrhage
            - infarct
            - infection
            - mass lesion
        - brain
            - haemorrhage
            - infarct
            - infection
            - tumour
        - systemic
            - organ failure
            - electrolytes
            - sepsis
            - toxins
- hemiparesis
    - acute stroke
        - ischaemic, haemorrhagic or subarachnoid haemorrhage
    - intracranial mass
    - meningitis/encephalitis
    - hypoglycaemia/hyperglycaemia
    - postictal Todd's paresis
    - hemiplegic migraine
    - Brown-Séquard syndrome
- quadriparesis/paraparesis +/- sensory level
    - spinal cord compression
    - spinal cord infarction
    - transverse myelitis
- proximal weakness
    - acute myopathy
    - Guillain-Barré syndrome (GBS)
    - diabetic lumbosacral radiculoplexus neuropathy (DLRN)
    - myasthenia gravis
    - Lambert-Eaton myasthenic syndrome (LEMS)
- distal weakness
    - vasculitic neuropathy
    - toxin-induced peripheral neuropathy

        - nerve compression syndromes
- assessment
    - relevant history
    - clinical examination
        - systemic features
        - neurological examination
    - investigations
        - bloods
            - glucose
            - electrolytes
            - calcium
            - magnesium
            - phosphate
            - U&Es
            - LFTs
            - coag
            - TFTs
            - CK
            - ESR
        - others according to suspected aetiology (e.g. CSF, vasculitis screen)
        - imaging (according to suspected aetiology)
            - CT brain (+/- contrast, venogram, angiogram)
            - MRI brain +/- angiography
            - CT or MRI spine
- neurological localisation based on physical examination
    - cerebral cortex, brainstem or spinal cord
        - weakness pattern
            - distal > proximal
            - extensors > flexors
            - hemiparesis or single limb
        - sensory loss
            - may be present depending on whether sensory tracts or cortex are involved
        - reflexes
            - increased (may be decreased initially)
        - acute causes
            - acute stroke
            - SAH
            - seizure
            - hypertensive encephalopathy
    - spinal cord
        - weakness pattern
            - distal > proximal
            - extensors > flexors
            - paraparesis
            - quadriparesis
            - rarely hemiparesis

        - reflexes
            - increased (may be decreased initially)
        - acute causes
            - epidural abscess
            - tumour
            - spinal cord infarct
    - anterior horn cell
        - weakness pattern
            - proximal and distal
            - fasciculations prominent
        - sensory loss
            - absent
        - reflexes
            - decreased if muscle bulk is severely decreased
            - increased in ALS
        - acute causes
            - ALS
            - polio
    - peripheral nerve
        - weakness pattern
            - in the distribution of the nerve, or diffusely present as stocking/glove weakness
        - sensory loss
            - present
        - reflexes
            - decreased
        - acute causes
            - Guillain-Barré syndrome
    - neuromuscular junction
        - weakness pattern
            - first in eye muscles, neck extensors, pharynx, diaphragm, followed by more generalised weakness
        - sensory loss
            - absent
        - reflexes
            - normal, decreased if muscle is paralysed
        - acute causes
            - botulism
            - tick bite
            - organophosphates
    - muscle
        - weakness
            - proximal
        - sensory loss
            - absent
        - reflexes
            - normal unless muscle severely weak

        - acute causes
            - rhabdomyolysis
    - UMN lesions may be difficult to distinguish from LMN lesions in the acute phase
        - acute UMN lesions may result in flaccid paralysis, normal or reduced tone and unreliable reflexes
        - acute LMN lesions may have insufficient time for atrophy to be evident and fasciculations are rarely seen
- management
    - airway and respiratory management
        - assess for intubation
        - key life threats are:
            - airway obstruction due to oropharyngeal collapse
            - respiratory failure due to diaphragmatic weakness
            - respiratory failure due to aspiration from inadequate airway protection
    - non-invasive ventilation
        - consider as a temporising measure in a neurologically stable patient with a neuromuscular condition expected to have a rapid resolution (e.g. myasthenia gravis exacerbation)
        - consider use for pre-oxygenation prior to intubation
    - avoid suxamethonium if there is evidence of underlying progressive neuromuscular disease (e.g. Guillain-Barré syndrome, chronic muscular weakness, prolonged immobility)
        - rocuronium (1.2mg/kg IBW IV) should be used for RSI
    - myasthenia gravis
        - use half dose of non-depolarising agents (e.g. rocuronium 0.6 mg/kg IBW IV)
    - if autonomic instability present or predicted
        - prior to intubation, prepare atropine/glycopyrronium, fluids and vasopressors
        - anticipate swings and avoid overshoot
    - specific therapy
        - according to underlying cause
    - supportive care and monitoring

NeuP9 dizziness and vertigo

categories of dizziness

- vertigo
    - an illusion of motion of either the subject or the environment
- presyncope
    - a feeling of impending faint or loss of consciousness
- disequilibrium
    - impaired balance and gait, may feel like there is a tilt to the floor
    - a sense of unsteadiness, not localised to the head, relieved by rest
    - most common cause is multiple sensory deficits in elderly people
- light-headedness
    - non-specific description of symptoms that cannot be identified as one of the above types

dizziness

- causes of dizziness/unsteadiness/lightheadedness
    - cardiovascular
        - postural hypotension
        - cerebrovascular disease
        - carotid sinus syndrome
        - vertebrobasilar insufficiency
        - aortic stenosis
        - subclavian steal syndrome
        - cardiac arrhythmias
    - neurological
        - following head injury
        - epilepsy
        - multiple sclerosis
        - Parkinsonism
        - dementia
        - brain tumours (especially brainstem and cerebellar)
        - idiopathic intracranial hypertension
        - normal pressure hydrocephalus
        - peripheral neuropathy (causing imbalance or unsteadiness)
    - otological
        - Ménière's disease
        - BPPV
        - vestibular neuritis and labyrinthitis
        - vestibular migraine
        - otosclerosis or Paget's disease of bone
        - middle ear trauma
        - following surgery (e.g. stapedectomy, cochlear implant)
        - tumours, cholesteatoma
    - metabolic
        - hypoglycaemia
        - adrenal insufficiency
        - hypothyroidism
    - haematological
        - anaemia
        - hyperviscosity
    - psychogenic
        - e.g. generalised anxiety, agoraphobia, panic attacks, hyperventilation
    - miscellaneous
        - viral illness
        - migraine
        - other infections
        - visual impairment
        - multisensory dizziness syndrome (reduced inputs from more than one sensory system
        - autoimmnune/connective tissue disorders
        - drug intoxication

        - iatrogenic (side effects of medication)
    - red flag signs of dizziness
        - abnormal neurological symptoms or signs
        - new headache
        - normal vestibulo-ocular reflex (head impulse test)
    - assessment
        - constant or episodic
        - triggered or spontaneous
            - e.g. triggered by movement, specific events, medication etc.
        - associated with other symptoms
            - e.g. hearing loss, headache, panic attacks, nausea, vomiting

vertigo

- background
    - affects 5% of adults per year
    - accounts for around 30% of dizzy patients in primary care
    - of all ED dizziness presentations, 43% were found to be due to a peripheral vestibular disorder
- definition
    - an illusion of rotatory movement
    - always implies an imbalance in the vestibular system but does not indicate where the imbalance originates
- pathophysiology
    - the ability of the body to maintain a sense of equilibrium relies on sensory inputs from the vestibular apparatus, visual system and proprioceptive stimuli from the neck and the rest of the body
    - the vestibular apparatus consists of the membranous labyrinth contained within the bony labyrinth lying in the petrous temporal bone, which connects via the vestibulocochlear nerve to the vestibular nuclei in the brainstem
        - the nuclei interconnect with neurones in the cerebellum, spinal cord and cerebral cortex
    - the membranous labyrinth consists of the three semicircular canals and two chambers, the saccule and utricle
        - flow of fluid (endolymph) in the canals stimulates cilia attached to a sensory organ located in the ampulla of each canal, the crista ampullaris
        - in the saccule and utricle, movement of calcified calcium carbonate crystals (statoconia or otoliths) stimulates cilia of another sensory organ, the macula
        - movement of the head in any plane modifies neural impulses transmitted via the vestibular nerve, connected to each of the sensory organs, to nuclei in the brainstem
    - vertigo results from an imbalance of either the received signals or information processing in the brainstem
    - nystagmus is an important clinical sign

- it consists of an initial smooth movement in one direction followed by a rapid movement (saccade) in the opposite direction
- it is described according to the direction of its fast component
- can be physiological or pathological
- a few self-limiting beats of nystagmus are normal on extreme lateral gaze

- history
  - may be subjective (I feel like I am moving/spinning) or objective (it feels like the world is moving/spinning)
  - peripheral vertigo
    - sudden onset
    - severe nausea and vomiting
    - worsened by position, often with a single critical position
    - associated auditory findings may be present
    - no associated neurological symptoms
  - central vertigo
    - occasionally very sudden onset but usually gradual
    - variable nausea and vomiting – usually limited systemic upset
    - little change with head position, associated with more than one position
    - associated auditory findings are rare
    - associated neurological symptoms are usually present
  - other useful history elements:
    - history of recurrent episodes
    - past history of vascular disease, hypertension or stroke (central cause more likely)
    - recent trauma or infection of the ear (peripheral cause more likely)
    - drugs associated with vertigo
      - e.g. ACEi, amiodarone, aminoglycosides, beta blockers, cocaine, phenytoin, salicylates, sildenafil
- examination
  - general assessment, including cardiovascular system
    - vertigo can be associated with vascular disease and atrial fibrillation
  - general neurological examination
    - cranial nerve or peripheral deficits associated with a cerebrovascular event or neoplastic lesion
      - e.g. cerebellopontine tumours may present with both vertigo and fifth and seventh nerve palsy
    - limb ataxia (abnormal finger-nose or heel-shin test) suggests cerebellar disease
  - ears
    - evidence of vesicles (Ramsay Hunt syndrome) or presence of tympanic perforation or cholesteatoma
  - hearing
    - gross testing with ticking watch/whispering/rubbing fingers together close to each ear
    - if reduction in hearing found, proceed to Weber's and Rinné's tests

- hearing loss or tinnitus almost always indicates a peripheral cause for vertigo
- HINTS examination
  - only valid in patients who have continuous ongoing vertigo at the time of assessment
  - differentiates between central and peripheral causes
  - head impulse
    - examiner faces patient and holds their head
    - patient fixates on tip of examiner's nose
    - head is rotated 20-40 degrees in each direction before being rapidly brought back to neutral
    - normal:
      - maintenance of continuous direction of gaze
    - central cause:
      - normal response
    - peripheral cause:
      - eye contact is lost and corrected with a saccade
  - nystagmus
    - patient is asked to look straight ahead, to the left and to the right whilst the direction of nystagmus is observed
    - central cause:
      - suggested by any vertical or rotational element or if the direction changes with direction of gaze
    - peripheral cause:
      - always horizontal
      - always has the fast phase in the same direction
      - often accentuated when the patient looks in the direction of the fast phase
  - test of skew
    - the patient faces the examiner and is asked to fixate on the tip of their nose
    - the eyes are alternately covered
    - central cause:
      - vertical alignment of the eyes may be different so a vertical corrective movement will be seen as the eye is covered and uncovered
    - peripheral cause:
      - no corrective movement

- acute vestibular neuritis
  - background
    - most common presentation of prolonged peripheral vertigo seen in the ED
    - typically in young and middle aged adults
    - may be caused by a viral infection, possible herpes simplex
    - caused by acute inflammation of the vestibular nerve
  - presentation
    - acute onset within minutes/hours

- exacerbated by movements of the head
- accompanying severe nausea and vomiting
- no other neurological deficit
- no disturbance of hearing (often found in other causes of peripheral vertigo)

- examination
  - spontaneous nystagmus in 2/3 patients, peripheral in character:
    - horizontal
    - unidirectional
    - suppressed by visual fixation
    - fast phase away from affected ear and most evident when looking to the unaffected side
- management
  - corticosteroids given acutely may improve longer term vestibular function

- cerebellar stroke
  - background
    - usually older patients with pre-existing risk factors (e.g. atrial fibrillation, diabetes)
  - presentation
    - onset is hyperacute within a few seconds
  - examination
    - vertigo is central in character:
      - unaffected by head position
      - little systemic upset
      - generally has co-existing neurological deficits (e.g. ataxia, depressed level of consciousness)
    - nystagmus is typically central:
      - horizontal, rotatory or vertical
      - bidirectional
      - not suppressed by visual fixation
- otomastoiditis
  - background
    - acute and chronic ear infection may directly infect or release toxins into the labyrinth causing acute labyrinthitis and peripheral vertigo
  - presentation
    - fever
    - ear pain
    - headache
    - hearing loss
  - examination
    - otoscopy reveals evidence of infection
- paroxysmal vertigo
  - without hearing loss
    - TIA
      - rarely presents as isolated central vertigo but can present with temporary features identical to cerebellar stroke

- benign positional paroxysmal vertigo (BPPV)
    - common, caused by dislocation of statoconia into the posterior semicirular canal
    - vertigo is related to changes in head position (e.g. turning over in bed or reaching upwards)
    - typically affects woman between 60 and 70
    - vertigo is peripheral and short lasting with fatiguing nystagmus in one direction
    - diagnosis is confirmed by the Hallpike manoeuvre and can be treated with the Epley manoeuvre, which can be self-administered once taught
        - Epley manoeuvre
            - treats BBPV due to posterior canal canaliths (?90% of cases)
            - recurrence rates may be as high as 50% over 5 years
            - patient lies flat with head hanging down over end of trolley (caution in elderly or neck pathology)
            - position held for 30 seconds and results in symptoms and nystagmus with the affected ear downmost
            - when nystagmus has stopped, rotate the patient's head to the opposite side (affected ear upwards) and hold for 30 seconds
            - the patient is then turned onto that side with their nose pointing to the floor for 30 seconds
            - finally, the patient is helped to a sitting position, keeping the head turned to the left
            - the process is repeated until nystagmus cannot be reproduced
- migraine
    - typically presents with episodic headaches accompanied by photophobia, nausea and vomiting
    - vertigo with either central or peripheral features can occur in up to 25% of patients

- with hearing loss
    - Ménière's disease
        - commonest cause of acute paroxysmal vertigo with hearing loss
        - caused by an increase in the pressure and volume of endolymph
        - patients normally present with an initial aura or aural fullness, followed by increasing tinnitus, fluctuating hearing loss and peripheral vertigo

        - clinical diagnosis, with investigations serving to rule out other diagnoses
        - management:
            - betahistine or diuretics are often prescribed, but evidence of efficacy is lacking
            - specialist treatments may include intratympanic gentamicin in selected cases
    - acoustic neuroma
        - vestibular schwannoma – a benign tumour of myelin-forming cells of the vestibulocochlear nerve
        - gradual progressive hearing loss and tinnitus are common symptoms
        - episodic central vertigo may also occur
- management
    - diagnosis
        - definitive diagnosis is not always possible in the ED
        - it is important to separate peripheral from central causes
    - central
        - all patients require further investigation as an in-patient or on an urgent outpatient basis
    - peripheral
        - most patients can be discharged home with arrangements for follow up with their GP or in an ENT clinic
        - some patients may require PT/OT input
        - some may require admission if unable to tolerate oral fluids even after treatment
    - treatment
        - vestibular suppressants
            - low dose benzodiazepines (diazepam 2mg, lorazepam 0.1mg)
            - anticholinergics (hyoscine)
            - antihistamines with anticholinergic and antiemetic properties (e.g. cinnarizine, cyclizine)
            - antiemetics such as prochlorperazine and metoclopramide are commonly used but may be associated with acute dystonic reactions
        - it is likely that recovery may be hampered by immobilisation and continued use of vestibular suppressants
        - physical therapies using vestibular exercises have been shown to significantly improve symptoms and function
            - patients can be advised to focus on an object while moving their head side to side then up and down
            - movements should be slow and slight to start with to prevent nausea, but can gradually be increased and repeated for several minutes 2-3 times a day

NeuC1 botulism

- mechanism:
    - endotoxins from Clostridium botulinum prevent release of acetylcholine at the neuromuscular junction
        - they are spore forming anaerobes with heat resistant spores
        - found in soil and marine sediment
- types:
    - food-borne
        - toxin absorbed from small intestine
            - often from home canned food
        - symptoms within 12-36 hours
        - GI distress, then neurological symptoms
        - parasympathetic dysfunction
            - dry mouth
            - blurred vision
            - dilated pupils
            - cardiovascular instability
        - severity proportional to amount of endotoxin
    - wound
        - from in vivo toxin production in abscessed devitalised tissue
        - 7 day incubation period
        - no GI prodrome
    - infant intestinal
        - ingestion of spores leads to colonisation of the GI tract and toxin production
        - first 2-4 months of life
        - constipation, poor feeding and lethargy followed by acute tetraparesis and respiratory failure
        - hypotonia
        - head lag
        - ptosis
        - reduced facial expression
        - reduced suck and swallow
        - loss of reflexes
        - slowly improves after weeks
    - adult intestinal
        - intestinal colonisation leads to endotoxin absorption when gastric barrier and intestinal flora are altered (e.g. surgery, gastric achlorhydria, broad spectrum antibiotics, inflammatory bowel disease
    - inhalational
        - bioweapon
    - iatrogenic
        - used to treat dystonia and spasticity or for cosmesis
- clinical features:
    - cranial neuropathies
        - diplopia, bulbar palsy
    - descending paralysis
    - bilateral symptoms

        - ataxia
    - no fever
    - no confusion
    - lack of sensory findings
- investigations:
    - collect source and samples (gastric, stool, serum)
    - LP
    - edrophonium test to rule out myasthenia gravis
    - EMG testing
- management:
    - notify public health
    - antitoxin
    - supportive care
        - may need intubation/ventilation
    - wound debridement and antibiotics for wound botulism

NeuC2 cerebral venous sinus thrombosis

- overview
    - thrombus formation in the deep or superficial venous drainage systems of the brain
    - multifactorial aetiology and variable presentation
    - rare (1% of all strokes)
    - more common in females (x3)
    - more common in younger adults (80% under 50)
    - diagnosis often delayed
    - goals of therapy are to stabilise the patient, remove the occlusion and stop clot propagation with anticoagulation
- pathophysiology
    - venous drainage of the brain
        - drained blood enters the dural sinuses (superior sagittal sinus, inferior sagittal sinus, lateral sinus, cavernous sinus and straight sinus, then the jugular vein
        - the superficial system has numerous anastomoses and predominantly drains the superior sagittal and lateral sinus
        - the deep white matter and basal ganglia are drained by the deep venous system towards the vein of Galen
    - cerebral venous thrombosis causes increased venous pressure
        - decreased CSF drainage and increased intracranial pressure
        - increased venule and capillary pressure
            - decreased capillary perfusion and subsequent cerebral perfusion causes ischaemia and cytotoxic oedema
            - disruption of blood brain barrier causes vasogenic oedema
            - venous and capillary rupture causes intraparenchymal haemorrhage
    - clinical presentation depends on:
        - thrombosis location
        - time since onset
        - presence of parenchymal brain involvement or intracranial hypertension
    - location of thrombus is associated with certain signs and symptoms

        - cavernous sinus thrombosis
            - ocular signs (orbital pain, chemosis, proptosis, oculomotor palsy)
        - cortical vein thrombosis
            - motor deficits, sensory deficits, seizures
        - sagittal sinus thrombosis
            - motor deficits, bilateral deficits, seizures
        - lateral sinus thrombosis
            - isolated intracranial hypertension
        - left transverse sinus thrombosis
            - aphasia
        - deep venous sinus thrombosis
            - behavioural symptoms
    - about 50% undergo haemorrhagic transformation prior to anticoagulation
- risk factors:
    - underlying thrombophilia (acquired or inherited)
    - high oestrogen exposure
        - oestrogen-based contraceptives
        - pregnancy
        - IVF treatment
    - other pro-thrombotic states
        - neoplastic disorders
            - e.g. direct compression, invasion of cerebral sinuses, chemotherapy
        - para-meningeal infections
            - e.g. sinusitis, otitis media, meningitis, head and neck infections
        - mechanical injury to sinuses or jugular veins
            - e.g. trauma, venous catheterisation, neurosurgical procedures
        - drugs
            - e.g. androgens, IV immunoglobulin
        - haematological disorders
            - e.g. paroxysmal nocturnal haemoglobinuria, myeloproliferative disorders
        - systemic disorders
            - e.g. SLE, Behçet's, inflammatory bowel disease, nephrotic syndrome
    - low CSF volume
- clinical presentation:
    - headache
        - no specific pattern
        - female in third trimester
        - female recently started on oral contraceptives
        - persistent and atypical in nature (gradual or sudden onset, localised or diffuse), especially in young adults
    - stroke
        - in absence of typical cardiovascular risk factors
        - may have motor, visual or sensory deficits
        - seizures are common
    - haemorrhagic infarcts on imaging that are multiple or do not conform to an arterial distribution

    - intracranial hypertension otherwise unexplained
        - may be insidious in onset with papilloedema or present as encephalopathy/coma
    - altered vision after recent sinusitis
    - neurological symptoms in patients with known risk factors for cerebral venous thrombosis
- investigations:
    - d-dimer
        - normal in 4% of cases and up to 40% of cases presenting with isolated headache
    - thrombophilia screen
        - 2-4 weeks after completing anticoagulation
    - imaging
        - CT venogram
            - 95% sensitive
            - can diagnose alternate differentials
        - MRI
            - avoids radiation and contrast problems
- management:
    - address life-threatening presentations
        - coma
        - seizures
        - raised ICP
    - specific therapy
        - anticoagulation
            - therapeutic heparin or LMWH
                - presence of intracranial haemorrhage with CVT is not a contraindication to anticoagulation
            - can transition to warfarin when stable
            - duration
                - 3-6 months if provoked (modifiable risk factor)
                - 6-12 months if unprovoked
                - indefinite if recurrent CVT, persistent por-thrombotic state or other thrombotic complications (e.g. PE)
        - thrombolysis (systemic or catheter-directed)
            - uncertain role
            - can consider as last resort if anticoagulation fails
        - early follow up CT or MR venogram if persisting or evolving symptoms to rule out thrombus propagation
    - supportive care and monitoring
        - adequate hydration
        - consider ICP monitoring if severe neurological deterioration
        - periodic assessment of visual fields and acuity
    - seek and treat underlying cause and complications
- prognosis:
    - 5% mortality during acute hospitalisation
    - good recovery in most, 88% of survivors have complete recovery or mild deficits

    - half have ongoing headaches
    - recurrence is rare
    - worse long term prognosis if:
        - cause is malignancy or CNS infection
        - associated intracranial haemorrhage
        - altered mental status or GCS <9 on admission
        - male
        - >37 years
        - CVT affects deep venous system

NeuC3 febrile convulsion

- definition:
    - seizure associated with fever in a young child
    - affects around 3% of children
    - most common between 6 months and 5 years
    - commonly from infection outside the CNS in a child who is otherwise neurologically normal
        - seizures due to CNS infection (meningitis, encephalitis) are not classified as febrile convulsions
    - fever is defined as >38°
- types:
    - simple febrile convulsion
        - isolated generalised tonic-clonic seizure lasting less than 15 minutes and not recurring within 24 hours or within the same febrile illness
    - complex febrile seizures
        - one or more of:
            - partial (focal) onset or focal feature during the seizure
            - duration of more than 15 minutes
            - recurrence within 24 hours or within the same febrile illness
            - incomplete recovery within 1 hour
    - febrile status epilepticus
        - febrile seizure lasting longer than 30 minutes
- assessment:
    - ABCDE assessment
    - history and examination to determine cause of fever
    - use of NICE traffic light system to determine likelihood of serious underlying illness
- indications for referral to paeds team:
    - age <18 months
    - signs of meningism
    - complex or prolonged seizures
    - no clear focus of infection
    - systemically unwell
    - parental anxiety
    - current or recurrent antibiotic use
    - first febrile convulsion (depends on place, some may observe overnight)
- guidance for parents:

- that the convulsion is not harmful to the child, does not cause brain damage and will not cause the child to die
- that febrile seizures are not the same as epilepsy
    - 1 in 50 chance of epilepsy developing later after 1 simple febrile convulsion
        - compared to 1 in131 for general population
    - no treatment available to reduce risk
- febrile seizure may recur
    - 1 in 3 will have another
- advice on managing future fevers
    - temperature control does not prevent recurrence but can make the child comfortable if distressed
        - aim is to ease symptoms and prevent dehydration
    - antipyretics should not be given just to reduce temperature
    - ensure adequate fluid intake
    - avoid tepid sponging, fanning and cold baths
- advice on management of recurrent febrile seizures
    - place child in recovery position
    - do not force anything into the child's mouth
    - note the time that the seizure starts and stay with the child
    - call for ambulance if seizure last >5 minutes
        - call GP if seizure resolves within 5 minutes
- explain that immunisations are still advised after a febrile seizure even if the seizure followed an immunisation

NeuC4 functional illness

- background
    - conditions arising primarily from a disorder of nervous system functioning rather than clearly identifiable pathophysiological disease
    - symptoms are genuine and may include:
        - paralysis
        - tremor
        - dystonia
        - sensory disturbance (including visual loss)
        - speech symptoms
        - seizures
    - second commonest reason for new neurology consultations
    - causes considerable physical disability and distress
    - should be diagnosed by someone with specific expertise in the diagnosis of neurological conditions
    - not a diagnosis of exclusion, should be positively diagnosed
    - often co-exists with other persistent physical symptoms such as dizziness, pain and fatigue
    - patients may also have other functional disorders such as irritable bowel syndrome, fibromyalgia or chronic pelvic pain
- history
    - list the symptoms
        - also ask about fatigue, pain, sleep disturbance and memory

    - describe a typical day
        - shows how disabled the person is
        - ask about good an bad days to assess variability
    - onset and course
        - particularly physical triggering such as injury, migraine or syncope
    - dissociative symptoms
        - e.g. depersonalisation (feeling disconnected from own body) and derealisation (feeling disconnected from the world)
        - patients may be relieved that their strange symptoms have a name
    - use of home video
        - can be helpful for episodic symptoms such as seizures or paroxysmal movement
    - ideas, concerns and expectations
    - stress and adverse life events
        - more common in FND but not always present
        - may help with individual formulation of aetiology and treatment plan but may also cause distress
        - can often wait until later visits
- clinical features
    - one or more of positive clinical features such as:
        - functional limb weakness
            - Hoover's sign
                - weakness of hip extension which returns transiently to normal during contralateral hip flexion against resistance – can be done sitting or lying
            - hip abductor sign
                - similar sign in relation to weakness of hup abduction that returns to normal with contralateral movement
        - functional movement disorders
            - functional tremor
                - evidence of distractibility with the 'entrainment test'
                - ask the patient to copy rhythmic movements of varying frequency made by the examiner between thumb and forefinger using one hand and then observe the response in the other hand
                - cessation of the tremor, 'entrainment' to the same rhythm or inability to copy the movement suggest functional tremor
        - functional dystonia
            - typically presents with a fixed position, usually clenched fist or inverted ankle
            - different to other types of dystonia, which are usually mobile
        - functional facial dystonia
            - usually presents with episodic contraction of platysma or orbicularis, resulting in a typical appearance (jaw deviation to one side)
        - functional or dissociative seizures
            - subjective descriptions often include:

                - symptoms of autonomic arousal such as palpitations, warmth, sweating
                - dissociative experiences (with or without fear)
                - often last seconds and are often not recalled
                - not diagnostic of functional seizures but can help to guide management (e.g. use of distraction techniques to gain control)
            - objective features
                - eyes tightly closed
                - tearfulness
                - duration more than 5 minutes
                - hyperventilation during a seizure
                - side to side head shaking
                - syncopal type presentation – sudden motionless unresponsiveness with eyes closed for more than 2 minutes
                - tongue biting and urinary incontinence may occur
        - functional visual loss
            - characteristically tubular (rather than conical) vision
                - visual field at 150cm is the same width as at 50cm
                - the diameter of a field should increase conically with distance
            - patients may also have visual field 'spiralling' on Goldmann perimetry with more constriction the longer the test goes on
- investigations
    - consider whether the patient could also have pathological disease
        - two diagnoses may be required
    - routine blood tests include B12 and TFTs are usually reasonable
    - if new symptoms occur it is important to consider whether they are likely to be related to the FND diagnosis
    - there is a danger of incidental findings in over-investigation and patients with a high clinical likelihood of FND should be warned in advance that tests are likely to be normal or have incidental findings
- diagnosis
    - common reasons for misdiagnosis include:
        - placing emphasis on psychological comorbidity
        - making judgements that symptoms are bizarre without considering whether they are typical of FND
        - relying on single signs rather than combinations of features
        - relying on normal investigations
    - common reasons for missing the diagnosis of FND include:
        - assuming it cannot be the diagnosis in a patient with no psychological comorbidity or prior functional disorder
        - patients who go against false stereotypes about functional disorders (e.g. patient who is male, older and working)
- explanation of diagnosis
    - patients should be provided with the name of the diagnosis and written information
    - support groups should be provided

    - demonstrating the clinical signs can be helpful (e.g. showing that the weak leg does go back to normal) – also demonstrates that it can be possible to retrain the brain
    - using the analogy of a problem with the software of the nervous system having a problem whilst the hardware of the brain is ok can be useful
- management
    - specific physiotherapy for functional motor symptoms
    - specialised cognitive behavioural therapy
    - multidisciplinary rehabilitation for refractory cases

NeuC5 Guillain-Barré

- pathophysiology:
    - acute infective polyneuropathy associated with demyelination of peripheral nerves
    - most common presentation is post-infection in otherwise healthy patient
        - 2/3 report symptoms of infection (GI or upper respiratory) within past 3 weeks
    - implicated organisms include:
        - Campylobacter jejuni
        - Haemophilus influenzae
        - Mycoplasma pneumoniae
        - Cytomegalovirus
        - Epstein-Barr virus
    - an immune activation event leads to autoantibody production
    - affects peripheral nerves, primarily spinal and cranial nerve roots
        - autonomic nerves sometimes affected
        - two pathologies of immune injury:
            - in demyelinating polyneuropathy the myelin sheath and Schwann cell components are targeted
            - in motor axonal neuropathy membranes on the nerve axon are targeted
- clinical features:
    - features:
        - symptoms usually move proximally (but not always)
        - progressive weakness in both arms and legs, often starting with the legs
        - reduced or absent tendon reflexes
        - relative symmetry of symptoms
        - progression up to 4 weeks
        - absence of fever at presentation
        - mild sensory symptoms or signs
        - facial weakness or other cranial nerve involvement
        - pain
            - muscular or radicular
            - commonly back pain and may precede weakness
- Miller-Fisher syndrome
    - variant of Guillain-Barré
    - cardinal features of ophthalmoplegia, often as part of a triad including ataxia and areflexia
    - may also have ptosis, facial or bulbar palsy along with mild limb weakness

- investigations:
    - LP
        - raised CSF protein (>0.4 g/L) with normal WCC
    - stool culture
        - for C. jejuni and others
    - ECG
        - risk of autonomic failure and cardiac arrhythmias
    - lung function tests
        - if any respiratory compromise or unable to walk
    - CK
        - if raised suggests alternative diagnosis such as myositis
    - electrolytes
        - exclude hypokalaemia and hypoglycaemia as symptom mimics
- management:
    - focus is on identification of possible cases
        - can have rapid progression from mild to respiratory compromise
    - serial lung function tests
        - FVC <20 ml/kg is an indication for intubation and ventilation
            - tracheostomy and slow wean often required
    - supportive treatment
    - plasma exchange or IV immunoglobulins in severe cases (e.g. unable to walk)
    - no proven benefit of steroids
- prognosis
    - recovery period lasts months to years
        - typically walking in 3 months for those who recover and full recovery in 6 months
    - can range from spontaneous recovery to permanent disability
        - 15% still have significant functional disability at 1 year
            - improvements can still occur after 3 or more years
    - poor prognosis with:
        - rapid onset of symptoms
        - increasing age
        - prior infection with C. jejuni
        - severe weakness
    - Erasmus GBS outcome scale (EGOS)
        - used at 2 weeks after admission to predict ability to walk at 6 months
        - based on:
            - age >40
            - preceding Campylobacter infection or diarrhoeal illness in past 4 weeks
            - high disability at nadir
    - Erasmus GBS respiratory insufficiency score (EGRIS)
        - predicts risk of respiratory failure
        - based on:
            - severity of weakness (MRC sum score)
            - onset of weakness (rapid)
            - facial palsy, bulbar weakness or both

NeuC6 meningitis and encephalitis

- bacterial meningitis
    - mainly affects babies, young children and adolescents
    - risk factors:
        - season (more common in winter)
        - exposure to smoke
        - recent influenza A infection
        - living in closed community
        - CSF leak
        - head and neck infections or surgery
        - immunosuppression
        - malnutrition
    - examination findings include:
        - meningism
            - head jolt accentuation test (lateral head rotation at about 2/second worsens headache)
                - very sensitive
        - papilloedema
        - tense fontanelle in babies
    - causes
        - meningococcal
            - commonest cause in UK
            - more prevalent in winter
            - clinical features:
                - acute onset
                - headache
                - nausea +/- vomiting
                - neck stiffness
                - focal neurological signs
                - altered level of consciousness
                - petechial rash in meningococcal septicaemia
            - morbidity and mortality
                - mortality 19-25% in meningococcal septicaemia
                - lowest risk of neurological sequelae compared to pneumococcal and H. influenzae
        - pneumococcal
            - second commonest in UK
            - clinical features:
                - as for meningococcal disease except no petechial rash
            - morbidity and mortality
                - 20% mortality
                - 40% have long term sequalae (e.g. deafness, seizures, functional impairment)
        - haemophilus
            - uncommon since introduction of Hib vaccine
            - clinical features:
                - as for meningococcal disease except no petechial rash

                - morbidity and mortality
                    - mortality rate <5%
                    - 20% have long term neurological sequelae
        - causes by age group:
            - neonate (<28 days)
                - Streptococcus agalactiae (Group B Strep)
                - E. coli
                - Strep pneumoniae
                - Listeria monocytogenes
            - infants
                - Neisseria meningitidis (meningococcus)
                - Strep pneumoniae (pneumococcus)
                - Haemophilus influenzae type b (Hib)
            - children
                - N. meningitidis
                - Strep pneumoniae
            - adults
                - Strep pneumoniae
                - N. meningitidis
                - Mycobacteria
        - complications:
            - intracranial
                - abscess
                - cerebritis
                - deafness
                - cognitive impairment
                - hydrocephalus
            - extracranial
                - septic shock
                - adrenal insufficiency from infarction (Waterhouse-Friederichsen syndrome)
                - acute renal failure
                - necrotising vacculitis
- viral meningitis
    - more common in summer
    - causes include:
        - enteroviruses (commonest)
        - HSV
        - VZV
        - mumps (rare since MMR)
        - no cause found in 50%
        - consider HIV and EBV in immunocompromised patients
        - clinical features:
            - subacute illness 1-7 days
            - high fever
            - headache
        - morbidity and mortality

- usually self-limiting
- HSV can progress to HSV encephalitis with increasing confusion, worsening fever
    - HSV encephalitis has 70% mortality rate

- cryptococcal meningitis
    - fungal infection caused by inhaling dust containing Cryptococcus neoformans
    - usually seen in immunocompromised patients
    - cryptococcal meningitis is an AIDS-defining illness
    - clinical features:
        - chronic or subacute symptoms
        - fever of unknown origin
        - chronic headaches
        - personality change
        - confusion
        - lethargy
    - morbidity and mortality
        - not curable in patients with AIDS
        - mortality rate 25-30%
        - 40% of survivors have significant neurological sequelae (e.g. loss of vision, cranial nerve palsies, hydrocephalus)
- tuberculous meningitis
    - usually in immunocompromised patients
    - patients who have not had a BCG vaccine are at greater risk
    - common secondary infection in patients with HIV
    - clinical features:
        - more indolent presentation
        - non-specific prodrome
            - headache, lethargy, photophobia, fever
        - most cases present within 2 weeks of symptoms onset
        - symptoms of meningism are rare in the immunocompromised
- encephalitis
    - inflammation of the brain parenchyma
    - clinical features:
        - fever
        - altered level of consciousness
        - headache
        - psychiatric symptoms
        - seizures
        - vomiting
        - focal neurological signs
        - memory loss
    - usually viral
    - causes include:
        - herpes simplex virus (most severe)
        - mumps
        - measles
        - varicella

        - rubella
        - adenovirus
        - CMV
        - Lyme disease
        - syphilis
        - TB
    - can also be autoimmune (e.g. acute disseminated encephalomyelitis – ADEM)
    - diagnosis (international encephalitis consortium):
        - major criterion (required):
            - presenting for medical attention with altered mental status defined as:
                - decreased or altered level of consciousness OR
                - lethargy OR
                - personality change AND
            - lasting ≥24 hours
        - minor criteria (2 for possible encephalitis, ≥3 for probable or confirmed encephalitis):
            - documented fever ≥38° within 72 hours before or after presentation
            - generalised or partial seizures not fully attributable to pre-existing seizure disorder
            - new onset of focal neurological findings
            - CSF WCC ≥5/mm^3
            - abnormality of brain parenchyma on neuroimaging suggestive of encephalitis that is either new from prior studies or appears acute
            - abnormality on EEG consistent with encephalitis and not attributable to another cause
            - AND
            - exclusion of encephalopathy caused by trauma, metabolic disturbance, tumour, alcohol abuse, sepsis and other non-infectious causes
    - management:
        - control seizures
        - acyclovir 10mg/kg TDS for 14 days
        - usually need to cover for possible bacterial meningitis with empirical antibiotics
    - prognosis worse with:
        - extremes of age (<1 year, >55 years)
        - comorbidities
        - immunodeficiency
        - virulent organism

NeuC7 multiple sclerosis

- pathophysiology
    - formation of multiple areas of scar tissue (sclerosis) along neurons
        - slows or blocks transmission of signals to and from brain and spinal cord
        - movement and sensation may be impaired
    - causes not completely understood

- autoimmune process with likely genetic and environmental factors
- patterns:
  - relapsing-remitting
    - symptoms come and go
    - remission periods followed by sudden relapses
    - 80% of people at onset
  - secondary progressive
    - follows on from relapsing-remitting
    - gradually more or worsening symptoms with fewer remissions
    - 50% of those with relapsing-remitting MS develop this during the first 10 years of illness
  - primary progressive
    - gradually developing and worsening symptoms from the beginning
    - 10-15% of people at onset
- clinically isolated syndrome is the typical first MS episode without clinical, radiological and immunological sign of any differential
  - delay before relapse (=conversion to clinically definite MS) can be several months up to more than a decade
- radiologically isolated syndrome is where asymptomatic patients have white matter anomalies suggestive of MS incidentally found
  - a third develop a clinical demyelinating event within 5 years

- epidemiology
  - prevalence 190 per 100,000
  - more than twice as common in females
  - highest prevalence is in the 60-69 age group
  - highest proportion of new female cases is in 30-34 and 40-44 age group; for males 45-49
- risk factors
  - family history
    - 2% with first degree relative with MS develop it
- diagnosis
  - clinical by consultant neurologist in most cases
  - McDonald criteria may be used to aid diagnosis
    - there is usually multiple relapses, dissemination in space and/or dissemination in time
      - dissemination in space
        - one or more MRI detected lesions typical of MS
        - further relapse showing damage to another part of the CNS
      - dissemination in time
        - oligoclonal bands OR
        - MRI evidence of new lesion since previous scan OR
        - further relapse
- presentation
  - visual
    - very common, usually due to demyelination of the optic nerve
    - can cause sight impairment or hemianopia
    - optic neuritis is a relatively common presenting symptom

        - acute, sometimes painful, reduction or loss of vision in one eye
    - eye movements
        - very common, may cause double vision
        - most frequent sign is symmetrical horizontal jerking nystagmus
        - lateral rectus weakness also common
    - facial weakness
        - Bell's palsy
        - trigeminal neuralgia
        - dysarthria
        - ataxia
        - other paroxysmal symptoms such as bursts of pain, bursts of paraesthesia, itching, cough, hiccup, painful spasm, complex gaze palsy
    - hearing and balance
        - deafness and feelings of unsteadiness are common
        - acute demyelination in brainstem causes severe positional vertigo, vomiting, ataxia and headache
    - cognitive symptoms
        - visual and auditory attention may be affected
        - effects on intelligence increase with duration and progression causing loss of memory more than language skills
    - psychological symptoms
        - depression is common
    - taste and smell
        - frequently found if specifically looked for
    - unpleasant sensations
        - tightness, burning, twisting, tearing and pulling sensations due to damage to posterior columns in the cervical cord
        - loss of thermal and pain sensation when spinothalamic tract involved
        - nonspecific tingling is common
    - paraesthesiae and numbness
        - loss of sensation in the legs ascending to the trunk when spinal nerves of dorsal or lumbar segments affected
        - may be sacral sparing but perineal and genital numbness with altered sphincter function are characteristic
    - transverse myelitis
        - acute episode of weakness or paralysis of both legs with sensory loss and loss of control of bowels and bladder – requires urgent hospital admission
    - autonomic system
        - bladder symptoms – urgency and frequency with incontinence
        - sexual problems – impotence in men, problems with spasticity , altered sensation and indwelling catheters
        - loss of thermoregulation – excessive sweating, pyrexia or hypothermia
    - other symptoms
        - include Horner's syndrome, abnormal cardiac rhythm, weight loss, SIADH
- investigations
    - exclude differentials with FBC, U&E, LFT, inflammatory markers, TFTs, glucose, HIV serology, calcium, B12
    - electrophysiology, MRI and LP for CSF likely to be required

- management
    - treat infections
    - liaise with specialist – may need course of steroid for relapse
    - DMARDs often used

NeuC8 myaesthenia gravis

- pathophysiology
    - antibody mediated autoimmune disruption of post-synaptic acetylcholine receptors at the neuromuscular junction
    - loss of up to 80% of functional receptors
    - typically young women
    - may have thymus hyperplasia
    - prevalence 14 per 100,000
- presentation
    - muscle fatiguability
        - may progress to permanent weakness
    - ptosis
    - commonly infected muscle groups include:
        - extra-ocular
        - bulbar
        - face
        - neck
    - reflexes usually normal but may be brisk
    - no sensory symptoms
    - symptoms most marked after prolonged exertion
        - including chewing fatigue
    - myasthenic crisis can occur:
        - upper airway muscle weakness leads to airway collapse and obstruction and inability to swallow secretions
- examination
    - swallow assessment
    - functional capacity
    - effectiveness of cough
    - jaw closure often weak and cannot be maintained against resistance
    - airway assessment
- investigations
    - edrophonium (Tensilon) test
    - electrophysiology studies/EMG
    - ACh receptor and muscle specific receptor tyrosine kinase antibody testing
    - CT mediastinum (looking for thymoma)
    - respiratory assessment
        - spirometry
        - PEFR
        - CXR
        - ABG
- management
    - cholinesterase inhibitors – pyridostigmine, rivastigmine

- plasma exchange
- IVIG
- corticosteroids for treatment resistant crises
- incentive spirometry and physiotherapy
- ICU referral:
    - VC <25ml/kg
    - weak cough
    - not clearing secretions
    - loss of airway protection
    - hypercapnic respiratory failure
    - pneumonia
    - planned or emergency surgical procedure
    - pregnancy with exacerbation
    - tapering of immunomodulatory therapy

NeuC9 Parkinson's disease and other movement disorders

- Parkinson's
    - degeneration of the dopaminergic pathways in the substantia nigra
    - idiopathic
    - Parkinsonism may be caused by drugs that block dopamine receptors or reduce storage of dopamine
        - may also follow encephalitis or exposure to certain toxins
    - epidemiology
        - second most common neurodegenerative disorder after Alzheimer's
        - typically develops between 55 and 65 years
        - male to female ratio 1.5:1
    - presentation
        - main features are tremor, rigidity and bradykinesia
            - tremor
                - 4-6 Hz
                - at rest
                - may be induced with concentration (e.g. reciting months backwards)
                - absent during activity
                - usually apparent in one limb or unilaterally before becoming generalised
            - rigidity
                - increase in resistance to passive movement
                - can produce characteristic flexed posture
                - may be increased by asking the patient to perform an action with the other limb
            - bradykinesia
                - slowness of voluntary movement and reduced automatic movements
                - reduced arm swing whilst walking
                - progressive reduction in amplitude of repeated movements (e.g. repeatedly opposing middle finger and thumb)

        - may still be able to move quickly in emergency situations
    - other features:
        - disturbed autonomic function with orthostatic hypotension, constipation, urinary disturbance
        - sleep disorders
        - neuropsychiatric symptoms
        - fixed facial expression with infrequent blinking
        - impaired swallowing +/- drooling
        - quiet voice
    - later features:
        - gait disturbance
            - difficulty in rising from sitting position and starting to walk
            - small shuffling steps with unsteadiness on turning and difficulty in stopping
            - tendency to fall
        - dementia
            - 2-6% increased risk compared to control population
- diagnosis
    - UK Parkinson's Disease Society (PDS) Brain Bank Criteria
        - diagnosis
            - bradykinesia and at least one of:
                - muscular rigidity
                - 4-6Hz resting tremor
                - postural instability not caused by primary visual, vestibular, cerebellar or proprioceptive dysfunction
        - exclusion criteria
            - history of repeated strokes with stepwise progression of Parkinsonian features
            - history of repeated head injury
            - history of definite encephalitis
            - oculogyric crisis
            - neuroleptic treatment at onset of symptoms
            - more than one affected relative
            - sustained remission
            - strictly unilateral features after three years
            - supranuclear gaze palsy
            - cerebellar signs
            - early severe autonomic involvement
            - early severe dementia with disturbance of memory, language and praxis
            - Babinski's sign
            - presence of a cerebral tumour or communicating hydrocephalus on CT scan
            - negative response to large doses of L-dopa (if malabsorption excluded)
            - exposure to MPTP (neurotoxin)

                - supportive prospective positive criteria of Parkinson's disease (3 or more required)
                    - unilateral onset
                    - rest tremor present
                    - progressive disorder
                    - persistent asymmetry affecting the side of onset most
                    - excellent response (70-100%) to L-dopa
                    - severe L-dopa induced chorea
                    - L-dopa response for five years or more
                    - clinical course of ten years or more
                    - hyposmia
                    - visual hallucinations
        - diagnosis
            - by a specialist
                - ideally within 6 weeks
            - CT or MRI if failure to respond to initial therapy
            - other investigations to exclude differentials such as Huntington's and Wilson's
        - complications
            - infections
            - aspiration pneumonia
            - bed sores
            - poor nutrition
            - falls
            - contractures
            - bowel and bladder disorders
        - prognosis
            - slow progression, mean duration 15 years
            - large variation, and early onset likely to have shorter lifespan
- other movement disorders
    - athetosis
        - sinuous, slow, involuntary writhing movements of fingers, hands, toes and feet
        - may involve arms, legs, neck and tongue
        - causes include:
            - asphyxia
            - neonatal jaundice
            - Huntington's
            - cerebrovascular disease
            - trauma
        - management includes:
            - medication (e.g. diazepam, haloperidol)
            - surgery
            - retraining techniques
    - chorea
        - continuous jerky movements
            - sudden, resulting posture is held for a few seconds

        - usually affects head, face or limbs
        - causes include:
            - adverse effects of medications
                - including for Parkinson's, epilepsy, schizophrenia
            - Huntington's
                - autosomal dominant, presents with chorea and dementia
                - motor, cognitive and psychiatric abnormalities
            - benign hereditary chorea
            - Wilson's disease
            - Sydenham's chorea (St Vitus's dance)
                - mainly associated with acute rheumatic fever
                - rare
                - children aged 7-12
                - behavioural disturbance followed by generalised chorea
                - recovery in 1-3 months
                - management with penicillin for rheumatic fever and diazepam, haloperidol or tetrabenazine for chorea
            - immune-mediated choreas
                - e.g. with SLE, antiphospholipid syndrome, vasculitis
            - infectious
                - e.g. meningitis, encephalitis, Lyme disease, prion disease
            - vascular
            - hormonal
                - e.g. hyperthyroidism, hyperparathyroidism with hypocalcaemia
            - pregnancy (chorea gravidarum)
            - metabolic (electrolyte disturbance)
            - vitamin B1 and B12 deficiency
            - paraneoplastic syndromes
            - other CNS conditions (e.g. trauma, intracranial tumour)
            - toxins (e.g. carbon monoxide, cyanide, alcohol, solvents, thallium)
- dystonias
    - sustained muscle contraction
        - frequently causes repetitive twisting movements or abnormal postures
        - severity can change depending on posture and voluntary activity
    - clinical diagnosis
    - treatments include oral medications, botulinum toxin and surgical procedures
    - primary dystonias
        - primary pure dystonia
            - mainly autosomal dominant
            - usually presents in children
        - blepharospasm
        - writer's cramp
    - secondary dystonias
        - hemidystonia

        - involves half of the body
        - usually secondary to structural lesion in basal ganglia
    - focal brain lesions
    - drugs
    - chemicals
    - Parkinson's disease
- hemiballism
  - wild flinging/throwing movements of one arm or leg
  - usually secondary to cerebrovascular event
  - vary from mild to severe and can cause injury
  - usually subside over 3-6 months
    - may require medication or neurosurgery to control them
- myoclonus
  - rapid muscle jerks
    - frequently repetitive
    - cause significant disability
  - types:
    - benign essential myoclonus
      - affects much of the body, up to 50 times/minute
      - presents in childhood or adolescence with mild disability
      - helped by alcohol and beta blockers
    - progressive myoclonic encephalopathies
      - as part of a range of other neurological disorders
    - static myoclonic encephalopathies
      - Lance-Adams syndrome after cerebral anoxia
    - myoclonic epilepsies
      - e.g. focal myoclonus restricted to one part of the body
  - management
    - usually requires a combination of drugs, e.g. antiepileptics, clonazepam, botulinum toxin
- spasmodic torticollis
  - twisting of the head and neck caused by shortened sternocleidomastoid muscle, tipping the head towards the shortened muscle and rotating the chin in the other direction
  - usually resolves in 24-48 hours, may last up to a week
  - management with analgesia, gentle exercise, heat/cold packs
  - check for evidence of head/neck infection
- tardive dyskinesia
  - usually follows at least six months of treatment with neuroleptics
    - risk is 5% per year of cumulative drug exposure
    - higher risk in older people
  - orofacial mouthing with lip-smacking and tongue protrusion, body rocking and distal chorea
- tics
  - repetitive stereotyped movements
  - can be voluntarily initiated and suppressed for short time
  - simple tics

                - sudden rapid twitch always occurring at same site
                - occurs in a quarter of children and resolves within a year
                - may persist into adulthood
            - complex multiple tics
                - more extensive and severe
                - may represent Tourette's, particularly when speaking/swearing involved
    - tremor
        - rhythmic movement of part of the body
        - most common types are essential tremor and Parkinsonian tremor
        - three types of pathological tremor:
            - static
                - in relaxed limb when fully at rest
                - causes include Parkinson's. Parkinsonism, other extrapyramidal disease, MS
            - postural
                - when a limb is static (can remain during movement)
                - includes physiological tremor, thyrotoxicosis, anxiety, alcohol, drugs, heavy metal poisoning, neurological disease, benign essential tremor, task-specific tremor
            - kinetic/action
                - during voluntary movement of an upper body part
                - intention tremor worsens as a goal-directed movement approaches its intended target
                - causes include brainstem or cerebellar disease such as MS, spinocerebellar degeneration, vascular disease, tumours
    - assessment:
        - Abnormal Involuntary Movement Scale can be used

NeuC10 peripheral neuropathy (acute)

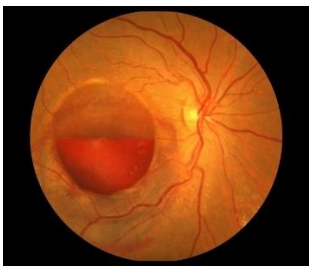
- types
    - predominantly motor, painful peripheral neuropathy, mononeuritis multiplex
        - mononeuritis multiplex is a painful, asynchronous sensory and motor peripheral neuropathy involving isolated damage to at least 2 separate nerve areas that can be in random areas of the body
    - causes of peripheral neuropathies (DAM IT BITCH)
        - **D**rugs and chemicals
            - e.g. lead, phenytoin, metronidazole, amiodarone, nitrofurantoin, organic solvents, carbon monoxide, organophosphates
        - **A**lcohol
            - with or without thiamine deficiency
        - **M**etabolic
            - diabetes, hypoglycaemia, uraemia
        - **I**nfection
            - HIV, leprosy, diphtheria, syphilis, post-infectious
        - **T**umour
            - paraneoplastic

        - **B**12 and other vitamin deficiencies
            - and pyridoxine excess
        - **I**diopathic and infiltrative (e.g. amyloidosis)
        - **T**oxins
            - e.g. botulism, ciguatera, tetrodotoxin
        - **C**onnective tissue disease
            - e.g. SLE, PAN, rheumatoid arthritis and congenital
        - **H**ypothyroidism
    - predominantly motor peripheral neuropathy
        - Guillain-Barré syndrome, chronic inflammatory polyradiculoneuropathy
        - hereditary motor and sensory neuropathy
        - diabetes
        - others – acute intermittent porphyria, lead poisoning, diphtheria, multifocal conduction block neuropathy
    - painful peripheral neuropathy (BADCAP)
        - **B**1 or B12 deficiency
        - **A**lcohol
        - **D**iabetes
        - **C**arcinoma
        - **A**rsenic or thallium poisoning
        - **P**orphyria
    - mononeuritis multiplex
        - acute
            - diabetes
            - polyarteritis nodosum
            - connective tissue diseases
        - chronic
            - multiple compressive neuropathies
            - sarcoidosis
            - acromegaly
            - leprosy
            - Lyme disease
            - idiopathic

NeuC11 subarachnoid haemorrhage

- definition
    - potentially fatal bleeding into the subarachnoid space, usually due to ruptured cerebral aneurysm
- pathophysiology
    - aneurysmal SAH
        - rupture of saccular aneurysm (60%)
        - rarely mycotic aneurysm
    - non-aneurysmal
        - perimesencephalic SAH (never associated with intraventricular extension)
        - AVM
        - angioma
        - neoplasm

        - cortical thrombosis
        - traumatic SAH
    - aneurysm sites – usually saccular or berry aneurysms
        - PCOM – 40%
        - ACOM – 35%
        - MCA – 20%
        - vertebrobasilar – 4%
- clinical features
    - sudden onset headache
        - classically occipital
        - unusual headache for patient
    - sentinel headache (40%)
    - transient or persistent loss of consciousness
    - vomiting
    - seizure or posturing
    - meningism
    - subhyaloid retinal haemorrhage or papilloedema on fundoscopy



https://imagebank.asrs.org/file/1059/subhyaloid-hemorrhage

    - neurological signs in keeping with the area near the aneurysm
    - risk factors
- risk factors
    - female (especially post-menopausal)
    - age >50
    - smoking
    - OCP
    - alcohol abuse
    - hypertension
    - connective tissue disorders (Marfan's, Ehlers-Danlos)
    - polycystic kidney disease
    - previous aneurysm or SAH
    - family history
    - coarctation of the aorta
    - fibromuscular dysplasia
- investigations
    - ECG
        - tall peaked T waves
        - ST depression
        - prolonged QT

        - arrhythmia
    - echo
        - neurogenic cardiomyopathy
    - bloods
        - hyponatraemia and hypovolaemia from SIADH or cerebral salt wasting – worsens vasospasm
        - raised troponin due to cardiomyopathy
    - LP
        - most sensitive for xanthochromia at 12 hours
    - imaging
        - CXR
            - neurogenic pulmonary oedema
        - CT head
            - 90% sensitive within 24 hours, 50% at 72 hours

https://radiopaedia.org/cases/aneurysmal-subarachnoid-haemorrhage-2

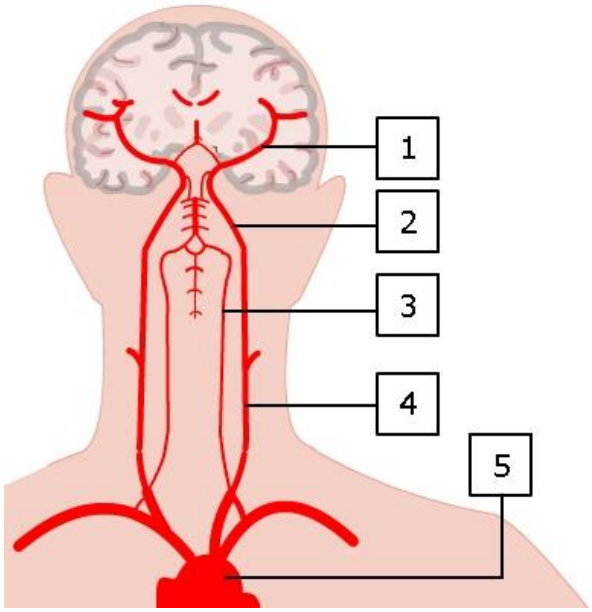
        - CTA
            - assess vascular anatomy
- grading
    - clinical
        - Hunt and Hess or WFNS
            - I – asymptomatic or mild headache/GCS 15, no motor deficit
            - II – moderate to severe headache, meningism, no weakness/GCS 13-14, no motor deficit
            - III – mild alteration in mental status, GCS 13-14, motor deficit
            - IV – hemiparesis/GCS 7-12 +/- motor deficit
            - V – posturing or comatose/GCS 3-6, motor deficit present or absent
        - CT (Fisher)
            - I – no blood
            - II – diffuse deposition of SAH without clots or layers of blood >1mm

        - III – localised clots and/or vertical layers of blood 1mm or more in thickness
        - IV – diffuse or no subarachnoid blood but intracerebral or intraventricular clots
- management:
    - elective intubation if airway not protected
    - routine monitoring
    - consider arterial line
    - anticonvulsants if seizure (not prophylactic)
    - dexamethasone sometimes used (no convincing evidence) to decrease oedema/irritation
    - isotonic fluids to maintain intravascular volume (watch for hyponatraemia)
    - manage hypertension with analgesia and antihypertensives
    - coiling if possible, otherwise clipping
- complications:
    - rebleeding
        - avoid hypertension
        - avoid decreases in ICP
        - sedation in the agitated
        - no coughing/Valsalva
        - avoid antifibrinolytics (decrease bleeding but increase vasospasm and hydrocephalus
- prognosis:
    - 25% die pre-hospital
    - worsens with increasing grade (I – 70% survival, V – 10% survival)
    - predictors of poor prognosis:
        - high grade
        - old age
        - co-morbidities
        - blood >1mm thick on CT
        - seizures
        - cerebral oedema
        - basilar artery aneurysm
        - symptomatic vasospasm
        - complications

NeuC12 stroke and TIA

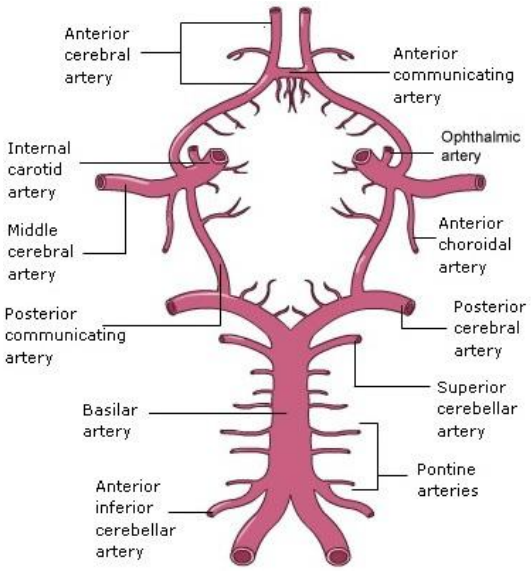
- background
    - strokes account for 11% of deaths in England and Wales
    - definition
        - an acute stroke is the clinical result of an interruption of the blood supply to a focal part of the brain causing loss of neurological function or death
    - approximately 85% are due to cerebral infarction (ischaemic stroke), 10% due to primary haemorrhage and 5% due to subarachnoid haemorrhage
- pathophysiology
    - aetiology of stroke and TIAs:
        - large artery atherothromboembolism (intracranial/extracranial)

        - small vessel disease
        - embolism from a cardiac source
        - other causes:
            - carotid or vertebral artery dissection
            - hypercoagulable states
            - sickle cell disease
            - 'undetermined' aetiology
    - ischaemic stroke
        - arterial occlusion leads to the abrupt cessation of distal blood flow
        - brain tissue receiving little or no blood flow is known as the ischaemic core and is made up of cells that die rapidly
            - typically 1.9 million neurons are lost every minute that the stroke is untreated
        - the ischaemic penumbra surrounds the ischaemic core
            - tissue is hypoperfused, functionally impaired and at risk of infarction, but may be saved if reperfused
            - it contains electrically excitable but viable cells
            - duration of ischaemia and absolute flow plays a crucial role; the longer it is untreated, the greater the risk of it undergoing infarction
    - vascular anatomy
- middle cerebral artery
    - the main branches of the internal carotids are the middle and anterior cerebral arteries (anterior circulation)
- internal carotid artery
    - the internal carotids supply blood to the anterior three fifths of the cerebrum:
        - frontal lobe
        - parietal lobe
        - part of the temporal lobe
- vertebral artery
    - the vertebrobasilar arteries supply the posterior two fifths of the cerebrum:
        - occipital lobe
        - part of the temporal lobe
    - they also supply part of the cerebellum and the brainstem (posterior circulation)
    - the basilar artery, formed by the union of the two vertebral arteries, gives off the posterior cerebral arteries
- common carotid artery
    - the common carotids each divide into two branches:
        - external carotid, supplying the exterior of the head, face and the greater part of the neck
        - internal carotid
- arch of the aorta
    - the portion between the ascending and descending aorta
    - the left common carotid originates directly off the highest part of the arch



https://www.rcemlearning.co.uk/reference/stroke-in-the-ed/#1629118206923-7b0ed505-9fcc

- circulation territories
  - the anterior and posterior circulations are linked via posterior communicating arteries forming the circle of Willis
  - the extent of each artery's territory varies between individuals, as does the presence or absence of collaterals
  - the signs of stroke depend on the site and size of the ischaemic injury
  - in anterior cerebral artery (ACA) occlusion, the leg will be more affected than the face and arm
  - in middle cerebral artery (MCA) occlusion, the face or arm is more affected than the leg



https://www.rcemlearning.co.uk/reference/stroke-in-the-ed/#1629118206923-7b0ed505-9fcc

- minor arteries

        - the major arteries give off small, penetrating vessels, which supply areas deep in the brain (sub-cortical areas) such as the internal capsule, thalamus and basal ganglia
        - strokes involving the small end arteries are called lacunar strokes
        - lacunar strokes involving the posterior limb of the internal capsule are typically associated with only motor signs, whereas those affecting the thalamus produce pure sensory signs
        - symptoms indicating cortical involvement include:
            - agnosia
            - dysphasia
            - sensory inattention
            - visual field deficits
- clinical assessment
    - FAST score
            - face weakness
            - arm weakness
            - speech disturbance
            - time
        - used by paramedics and in public awareness campaigns
    - ED procedures
        - rapid structured assessment
        - exclusion of hypoglycaemia
    - ROSIER (Recognition Of Stroke In the Emergency Room)
        - score is from -2 to +5
            - clinical history (-1 for each)
                - loss of consciousness
                - convulsive fit
            - neurological signs (+1 for each)
                - face weakness
                - arm weakness
                - leg weakness
                - speech disturbance
                - visual field defect
        - a score of 1 or above makes stroke more likely (PPV 90%, NPV 88%)
        - if the score is negative, a stroke mimic should be considered
- stroke mimics
    - migraine
        - often associated with an aura (positive symptom)
            - a stroke involves loss of neurological function (negative symptoms)
        - headache is not a feature of ischaemic stroke but is often associated with intracerebral haemorrhage
    - hypoglycaemia
        - identified with bedside glucose test
        - all thrombolysis protocols require exclusion of hypoglycaemia
    - seizure
        - may be a complication of an acute stroke or may develop in someone with a history of stroke

        - presentation with a seizure reduces the odds ratio of the patient having a stroke (OR 0.28)
    - brain tumour, space occupying lesion or subdural
        - usually more gradual onset, though features may be the same
        - rapidly distinguished on brain imaging
    - sepsis
        - patient usually has systemic features such as fever
        - severe sepsis associated with systemic hypoperfusion may cause watershed area neurological dysfunction
    - syncope
        - stroke rarely presents with syncope alone
    - toxic metabolic states
        - hyperglycaemia and hyponatraemia can present with focal neurology
        - confusion and slurred speech may also be present
    - CN VII nerve palsy
        - a peripheral CN VII is a lower motor neuron lesion so the whole of one side of the face is weak
        - in an upper motor neuron lesion from a stroke (MCA territory) only the lower two thirds of the face is weak
- investigations
    - non-contrast CT
        - main aim is to exclude a bleed as the cause of focal neurological signs
        - early signs of ischaemia may or may not be seen (e.g. loss of differentiation of grey white matter interface)
        - CT may also show another cause such as brain tumour or subdural
        - NICE guidelines
            - brain imaging should be performed immediately (ideally the next slot and definitely within an hour, whichever is sooner) for people with acute stroke if any of the following apply:
                - indications for thrombolysis or early anticoagulation treatment
                - on anticoagulation treatment
                - known bleeding tendency
                - depressed level of consciousness (GCS <13)
                - unexplained progressive or fluctuating symptoms
                - papilloedema, neck stiffness or fever
                - severe headache at onset of stroke systems
            - for people without these features, scanning should be done as soon as possible, and within a maximum of 24 hours after the onset of symptoms
        - MCA sign/loss of insular ribbon
            - the hyperdense MCA sign refers to the appearance of the middle cerebral artery on CT; it has been associated with poor outcome
            - there is increased attenuation of the proximal portion of the MCA, it is one of the early signs of an MCA infarct
            - the sign is typically seen within 90 minutes of the ischaemic event and is therefore very important to recognise

        - it has around 100% sensitivity but only 30% specificity
      - usually associated with the insular ribbon sign – loss of definition of the grey-white interface in the lateral margin of the insular cortex
  - MRI
    - higher sensitivity for early ischaemic changes and can image the posterior fossa more reliably
    - with diffusion weighted MRI, diffusion perfusion mismatch may show the area of potentially reversible ischaemia and better identify patients beyond current guidelines who would benefit from thrombolytic therapy
    - availability can be limited
  - other tests
    - bloods
      - FBC
      - U&E
      - coagulation studies
      - blood glucose
    - ECG
    - CXR
  - for selected ED patients:
    - toxicology
    - pregnancy test
    - LFTs
- management
  - aims
    - restoration of brain perfusion to reduce the effects of the primary insult and prevent secondary brain injury
    - effective stroke care, multi-disciplinary involving the stroke team, radiology and the stroke unit
    - thrombolysis – patients presenting within a few hours may benefit from rtPA (tissue plasminogen activator)
      - a focussed medical history including exact time of onset is required
      - eligibility for thrombolysis was extended to 4.5 hours following publication of the European Cooperative Acute Stroke Study
  - ABC management
    - airway and breathing
      - positioning (e.g. if patient slumping and obstructing airway) or intubation (prevention of secondary injury) may be required
      - oxygen if required to maintain sats 94-98%
      - nil by mouth until swallow has been assessed
  - circulation
    - patients may be dehydrated (e.g. after a long lie or if unable to swallow)
    - poorer outcomes are associated with a systolic <100 or diastolic <70
    - boluses of 250-500ml of saline can be used with the aim of euvolaemia and avoidance of fluid overload which may exacerbate cerebral oedema
    - hypertension should rarely be treated in the acute phase; reasons for doing so include:
      - hypertensive encephalopathy

                - hypertensive nephropathy
                - hypertensive cardiac failure/myocardial infarction
                - aortic dissection
                - pre-eclampsia/eclampsia
                - intracerebral haemorrhage with systolic over 200
            - in patients being considered for thrombolysis the target pressure is less than185/110
    - pyrexia
        - associated with increased morbidity and mortality
            - no trials have been done to see if treating it helps
    - hyperglycaemia
        - associated with poor outcomes after ischaemic stroke, including in patients treated with thrombolytic agents
            - there is an increased rate of intra-cerebral haemorrhage
        - evidence for improvement with treatment of hyperglycaemia is unclear
            - current consensus is to aim for a glucose of 5-15 mmol/l, which may require an insulin infusion
    - focussed neurological examination
        - most widely adopted tool is the National Institutes of Health Stroke Scale (NIHSS)
            - used to provide an objective baseline assessment of neurological function
    - thrombolysis
        - NICE recommends alteplase used by experienced clinicians
            - protocols and training should be in place
        - by current NICE guidelines around 20% of patients are eligible to receive thrombolysis treatment
        - communication with patients
            - risks and benefits should be discussed with patients and relatives and written information provided
            - numbers needed to treat
                - disability free survival if within 3 hours: 10
                - disability free survival if within 4.5 hours: 20
            - numbers needed to harm
                - to do worse: 20
                - to kill or leave permanently disabled: 50
        - indications for consideration of thrombolysis:
            - clinical signs and symptoms consistent with acute stroke
            - clear time of onset
            - presentation within 4.5 hours of onset
            - no contra-indications
            - individualised risk: benefit assessment favouring thrombolysis
        - contra-indications for thrombolysis:
            - imaging
                - intracranial haemorrhage
                - greater than one third middle cerebral artery territory acute ischaemic change (i.e. very severe stroke)

        - extensive small vessel disease
    - history
        - symptoms began more than 4.5 hours earlier or unknown time of onset
        - patient already receiving effective oral anticoagulation treatment
        - seizure at stroke onset
        - history of stroke or head injury in the past three months
        - history of any previous stroke and concomitant diabetes
        - history of any previous intracranial haemorrhage
        - history consistent with subarachnoid haemorrhage (even if CT is normal)
        - history of previous major surgery or significant trauma in the last 3 months
        - previous CNS damage (including spinal/intracranial surgery, neoplasm or aneurysm)
        - history of gastrointestinal or urinary tract haemorrhage within 21 days or documented ulcerative gastrointestinal disease in the last 3 months
        - recent (less than 10 days) arterial puncture at a non-compressible site (subclavian/jugular)
        - recent (less than 10 days) traumatic external heart massage or obstetrical delivery
        - recent lumbar puncture
        - heparin treatment within last two days
        - severe liver disease, acute pancreatitis, neoplasm with increased bleeding risk
        - significant bleeding disorder at present or within the past 6 months, or know haemorrhagic diathesis
    - investigations
        - platelets <100
        - INR >1.7/ elevated thromboplastin time
        - blood glucose <50 or >400 mg/dl
    - examination
        - symptoms rapidly improving
        - low NIHSS score (four or less) or very high NIHSS score (25 or more)
        - systolic BP consistently >185
        - diastolic BP consistently >110
- reperfusion therapy and NOACs
    - the Royal College of Physicians recommends that patients taking a NOAC are excluded from receiving alteplase
    - the exception is dabigatran – if PT and APTT are both normal thrombolysis can be considered
    - use of reversal agents then alteplase is not recommended
- medication

                - recommended medication is alteplase at 0.9mg/kg (max 90mg) as an infusion over 60 minutes, with the first 10% of the total being given as a bolus
            - time targets (US guidelines)
                - door to physician – 10 minutes
                - door to stroke team – 15 minutes
                - door to CT initiation – 25 minutes
                - door to CT interpretation – 45 minutes
                - door to drug (80% compliance) – 60 minutes
                - door to stroke unit admission – 3 hours
        - other treatments
            - aspirin
                - should be withheld for 24 hours in patients treated with tPA
                - otherwise, 300mg should be given as soon as haemorrhage has been excluded
                    - oral, rectal, NG options
                    - if the patient previously had dyspepsia with aspirin, a PPI should be co-administered
                    - if the patient is allergic or intolerant, an alternative antiplatelet (e.g. clopidogrel) should be given
        - endovascular therapy
            - mechanical thrombectomy – offers additional benefit as an adjunct to thrombolysis in certain patient populations
            - UK Royal College of Physicians recommend it for:
                - patients with a proximal intracranial large vessel occlusion causing a disabling neurological deficit (NIHSS score of 6 or more) if the procedure can begin (arterial puncture) within 5 hours of onset
                - if the large artery occlusion is in the posterior circulation, treatment up to 24 hours after onset may be appropriate
            - the proximal large vessel occlusions include the intracranial or extracranial ICA, M1 or M2 middle cerebral artery or basilar artery
            - access to the service is challenging
- disposition
    - stroke patients receiving inpatient care on a stroke unit are more likely to be alive, independent and living at home one year after the stroke
    - some patients may be treated at a regional centre/hyperacute centre then transferred back to their local hospital after 72 hours for rehabilitation
- specific stroke syndromes
    - malignant middle cerebral artery infarct
        - large space-occupying middle cerebral artery or hemispheric ischaemic brain infarcts are associated with the development of massive brain oedema, which may lead to herniation and early death
        - associated with 80% mortality due to herniation during the first week
        - about 5% of strokes result in this complication, almost all caused by embolic occlusion of the proximal middle cerebral artery
        - the patient's condition usually deteriorates on the third to fifth day despite maximal conservative ICU treatment

        - young patients are particularly at risk due to having little cerebral atrophy so brain swelling causes significant symptoms
        - hemicraniectomy relieves mechanical compression and may improve cerebral perfusion and prevent further ischaemia
            - if indicated, it should be performed within 48 hours of stroke onset
        - UK National guidelines
            - patients with MCA infarction who meet all the criteria should be considered for compressive hemicraniectomy:
                - pre-stroke modified Rankin Scale score of less than 2
                - clinical deficits indicating infarction in the territory of the MCA
                - NIHSS score of more than 15
                - decrease in level of consciousness to a score of 1 or more on item 1a of the NIHSS (i.e. no longer alert)
                - signs on CT of at least 50% of the MCA territory with or without additional infarction in the territory of the anterior or posterior cerebral artery on the same side, or infarct volume greater than 145 cubic cm on diffusion-weighted MRI
            - patients should be referred to neurosurgery within 24 hours of stroke onset and treated within 48 hours of stroke onset
            - neurosurgical centres may not have much experience of it and senior decision-maker involvement may be required
    - basilar artery stroke
        - rare but under-recognised
        - high mortality and morbidity rate
        - the basilar artery is formed by the union of the two vertebral arteries and terminates as the posterior cerebral arteries
        - signs and symptoms depend on where the occlusion is
        - patients will frequently have had preceding posterior circulation TIA symptoms
        - there may be:
            - basilar artery occlusion that can cause severe quadriplegia, coma and locked-in syndrome
            - a stuttering course of posterior circulation symptoms that finally become progressively disabling; the most characteristic motor manifestation is an asymmetric quadriparesis
        - bulbar symptoms include:
            - facial weakness
            - jaw weakness
            - dysarthria
            - dysphonia
            - dysphagia
            - eye signs (abnormalities of oculomotor and pupillary function)
        - diagnosis
            - CT angiography
        - treatment

- patients have a poor outcome with conservative treatment
- IV or preferably intra-arterial thrombolysis or mechanical thrombectomy may be beneficial

- carotid artery dissection
  - important cause of stroke to consider in patients younger than 50
  - has an associated mortality of up to 5%
  - incidence is around 3 per 100,000 population, but likely higher as often goes undiagnosed
  - risk factors include:
    - head or neck manipulation or trauma
    - aneurysm
    - hypertension
    - atherosclerosis
    - dissection should be considered in patients with ischaemic stroke without the usual risk factors
  - presentation
    - incidental finding of atraumatic disease
    - cerebrovascular events
    - ipsilateral headache
    - face or neck pain
    - Horner's syndrome
    - 25% can have neck pain alone, usually sudden, severe and persistent
  - diagnosis
    - requires immediate CTA or MRA
  - management
    - full resolution occurs in >90%
- cervical artery dissection
  - background
    - cause around 13% of ischaemic strokes in patients under 45
    - easily missed, high index of suspicion required
    - encompasses dissections of both the carotid and vertebral arteries
      - similar aetiology but different presentations
    - carotid artery dissection is twice as common as vertebral artery dissection
  - pathophysiology
    - the basic lesion is an intramural haematoma in the vessel wall
      - may arise from a haemorrhage in the vaso vasorum
      - can be the result of a tear in the intima of the vessel causing an intimal flap
    - the intramural haematoma can narrow the vessel lumen or cause an aneurysmal dilatation
    - clinical features occur through two mechanisms:
      - low flow through a stenosed vessel and intimal damage can cause thrombus formation leading to neurological sequalae
      - the haematoma, with or without aneurysm formation, can cause symptoms from compression or adjacent structures)
  - risk factors include:
    - trauma

            - may be trivial, even sneezing or yoga
        - respiratory tract infection
        - fibromuscular dysplasia
        - cystic medial necrosis
        - Marfan syndrome
        - Ehlers-Danlos syndrome
    - clinical presentation
        - initial symptom is usually pain
            - headache in 70%
            - in vertebral artery dissection it is often occipital pain extending into the neck
            - in carotid dissection it can be headache, neck pain or facial pain
            - the pain is on the same side as the dissection
        - focal neurological signs follow in around 60% of patients
            - often develop several days after the onset of pain
            - display different patterns depending on the vessel involved
    - vertebral artery dissection
        - affects the posterior circulation
        - most often presents as a lateral medullary syndrome due to emboli in the posterior inferior cerebellar artery
        - this causes:
            - ipsilateral facial pain and temperature sensory loss (spinal trigeminal nucleus)
            - contralateral pain and temperature sensory loss from the body (lateral spinothalamic tract after decussation)
            - dysphagia and dysarthria (nucleus ambiguus motor fibres of CN IX and X)
            - ataxia (cerebellar peduncle)
            - vertigo (vestibular nuclei)
        - patients may also have a Horner's syndrome caused by ischaemia of the of the descending sympathetic fibres
    - carotid artery dissection
        - may result in focal weakness and/or sensory loss and amaurosis fugax
        - there may be a carotid bruit
        - can cause a Horner's syndrome due to compression of the sympathetic fibres travelling with the internal carotid
            - sweating is preserved as it is regulated by sympathetic fibres in the external carotid plexus
    - differential
        - varies according to presentation and presence/absence of trauma
        - may include:
            - migraine
            - stroke
            - TIA
            - subarachnoid haemorrhage
            - traumatic brain injury
    - investigation

        - non-contrast CT
            - first line investigation
            - may be normal and can falsely reassure unless there is a high degree of clinical suspicion
        - CT angiography
            - may provide a higher yield if there is suspicion of dissection
            - may also be useful in trauma patients undergoing CT for other indications
            - MR angiography is the investigation of choice in the UK
        - doppler ultrasound
            - demonstrates abnormal flow in around 90% of patients with either vertebral or carotid artery dissection
                - technically difficult to scan the distal internal carotid
    - management
        - mainstay of treatment is anticoagulation
            - use is only supported by case reports and not evidence based
            - makes sense as the neurological sequelae are caused by thromboembolism
            - haemorrhage must be excluded first with CT brain
        - endovascular stenting
            - particularly an option for patients with a contraindication to anticoagulation
    - prognosis
        - mortality is around 5%
        - 75-80% of those surviving make a full recovery

NeuC13 tetanus

- background
    - immunisation was introduced in the 1950s, since when acute tetanus has become rare in the industrialised world
    - there were over 16,000 reported cases in 2008, with actual levels likely to be much higher
    - most UK cases are in over 45s who may not be adequately immunised
        - there is an average of around 10 cases/year in the UK
        - overall case fatality around 29%
    - tetanus is a notifiable disease in the UK
- definition
    - four patterns of disease:
        - generalised tetanus
            - mild to moderate trismus and one or more of:
                - spasticity
                - dysphagia
                - respiratory problems
                - spasms
                - autonomic dysfunction
        - localised tetanus

                - increased rigidity and spasticity of muscle groups adjacent to the site of the infection
                - good prognosis if remaining localised but may progress to generalised tetanus
            - cephalic tetanus
                - presents as isolated cranial nerve palsies
                - usually develops from chronic middle ear infections or head wounds
                - left untreated, progresses to generalised tetanus
            - neonatal tetanus
                - significant cause of neonatal death in the developing world due to unvaccinated mothers and poor cord hygiene
                - presents a few days after birth with general irritability, poor sucking, grimacing, generalised rigidity and spasms
- pathophysiology
    - caused by clostridium tetani
        - obligate anaerobic Gram-positive bacillus
        - bacillus forms resistant spores which are widespread in soil and animal and human faeces
    - spores may enter via acute wounds, leg ulcers, burns, abdominal surgery or IV drug use
    - wounds may be trivial and there is no recognised wound in up to 25% of cases
    - cases related to IV drug use are due to contamination of the drug itself and may result in localised outbreaks
    - incubation period is 4-14 days in 75% of cases
        - may be as little as 1 day
    - two toxins are released by the bacilli:
        - tetanolysin
            - causes local tissue damage, optimising conditions for the anaerobic organisms
        - tetanospasmin
            - enters nerve endings at the neuromuscular junction by infiltration from infected muscle or systemically via vascular and lymphatic routes
            - intraneural migration to the neurones of the spinal cord follows, where it exerts its neurotoxic effect
            - within the spinal cord, tetanospasmin blocks release of inhibitory neurotransmitters, particularly gamma-aminobutyric acid (GABA) and glycine
                - this reduces suppression of motor reflexes and the resulting exaggerated response to sensory stimuli produces the spasms typical of tetanus
            - spasms may cause dysphagia, respiratory distress (quick shallow breaths), poor suckling in infants, abdominal rigidity, long bone fractures and joint dislocations
            - tetanospasmin also inhibits activation of motor end-plates, causing generalised muscular weakness and respiratory failure

- it also affects neurones of the autonomic nervous system with sympathetic overactivity
- if the distance from the entry point to the CNS is shorter, the symptom onset of generalised tetanus will be quicker, hence the early onset of such symptoms in head and neck wound related cases
- intraneural toxin cannot be neutralised, so a patient's condition may continue to deteriorate after the onset of treatment
    - recovery requires new synapse formation which takes 2-4 months

- presentation
    - 75% of patients with generalised tetanus present with trismus
    - the other classic feature is facial muscle involvement (risus sardonicus)
    - within days, muscle spasm and rigidity spread distally, affecting trunk and limb muscles
    - pharyngeal muscle involvement may cause a gradual onset of dysphagia
    - laryngospasm may occur
    - most patients develop both spasticity and episodic spasms provoked by minor sensory stimulus
        - typical spasms include opisthotonos, fist clenching and arm flexion and leg extension
    - spasms may cause respiratory compromise, joint dislocations, fractures and rhabdomyolysis
    - patients remain conscious during these episodes, which are extremely painful
    - autonomic nervous system involvement
        - occurs several days after the onset of spasms
        - features:
            - blood pressure instability
            - tachyarrhythmias, bradyarrhythmias, asystole
            - sweating, hypersalivation and hyperthermia
            - urinary retention and high-output renal failure
    - sudden cardiac death is the most frequent cause of death in ventilated patients
    - abdominal pain and rigidity may mimic an acute abdomen
        - constipation, diarrhoea and ileus may also occur
    - pharyngeal and laryngeal spasm may cause airway obstruction
    - risk of aspiration is increased due to:
        - poor swallow
        - increased saliva
        - inadequate cough
        - raised intra-abdominal pressure
    - pneumonia is common
- diagnosis
    - spatula test
        - touching the oropharynx in normal patients results in a gag reflex and efforts to expel the spatula (negative result)
        - if tetanus is present, reflex spasm of the masseters occurs and the patient bites the spatula (positive result)
        - test is very specific and highly sensitive

- differential
    - orofacial infection
    - quinsy
    - dystonic drug reactions
    - atypical seizures
    - hypocalcaemia
    - strychnine poisoning
    - temporomandibular joint disorders
- investigations
    - tetanus toxin in a serum sample
        - a positive result confirms the diagnosis
        - a negative result does not exclude it
    - the sample must be taken prior to the administration of immunoglobulin
    - it is rare to get a positive a positive result from a wound site
    - measuring tetanus antibodies in serum:
        - absent or low levels of antibodies suggests absent or inadequate immunity and indirectly supports a diagnosis of tetanus
- prognosis
    - overall mortality is around 20% in the developed world
    - mortality is 30% for those needing ventilation and 4% for those who do not
    - age affects outcome, with mortality of 60% for those over 60 compared to 8% for those aged 20-59
    - predictors of more severe illness
        - short incubation period
        - delay in treatment
        - early onset convulsions
        - cephalic and neonatal tetanus have a much worse prognosis
            - mortality exceeds 70% for neonatal tetanus
    - tetanus which remains localised has a good prognosis
- grading of severity (by Ablett, used by the Health Protection Agency)
    - grade 1 (mild)
        - mild to moderate trismus and general spasticity
        - little or no dysphagia
        - no respiratory embarrassment
    - grade 2 (moderate)
        - moderate trismus and general spasticity
        - some dysphagia and respiratory embarrassment
        - fleeting spasms
    - grade 3a (severe)
        - severe trismus and general spasticity
        - severe dysphagia and respiratory difficulty
        - severe prolonged spasms (both spontaneous and on stimulation)
    - grade 3b (very severe)
        - as for severe plus autonomic dysfunction, particularly sympathetic overdrive
- vaccination
    - a full course of vaccination does not guarantee immunity
    - partial vaccination offers some protection and results in less severe disease

- recovery
    - patients who survive make a full recovery in 2-4 months
    - occasionally, patients are left with residual weakness and hypotonia
- management
    - initial management
        - environment
            - quiet environment to minimise external stimulation and resultant spasms
        - ABCDE assessment
            - provide support as needed
        - vital signs
            - should be monitored
        - IV access
            - routine bloods plus CK
            - spare serum sample to test for tetanus toxin or serology later
        - muscle spasms
            - diazepam is the most common treatment
                - there is inadequate evidence for this but theoretical benefits as a GABA agonist
            - if diazepam is not sufficient or respiratory depression too great, neuromuscular blockade and ventilation may be required
            - other agents may be used (little or no evidence base to distinguish one from another):
                - phenobarbitone
                - chlorpromazine
                - dantrolene
                - propofol (non-anaesthetic dose)
                - intrathecal baclofen
    - secondary management
        - autonomic dysfunction
            - most effective treatment is unclear – most evidence is from case studies or small samples
            - treatments have included verapamil and atropine
            - morphine seems to be useful to control cardiovascular instability
            - beta blockade has not been successful, with increased rates of profound hypotension and sudden death
            - magnesium sulphate has been shown to reduce autonomic instability but with no effect on survival
        - wounds
            - should be debrided to remove the reservoir of tetanus bacilli
            - surgery should be delayed for several hours after administration of TIG as it may release tetanus toxin from the wound site
            - removal of 2cm of surrounding viable tissue is recommended
        - antibiotics
            - should be given
            - traditionally penicillin, but this is a GABA antagonist and may aggravate rigidity and muscle spasms

                - metronidazole is effective and probably the drug of choice
                - alternatives include erythromycin, tetracycline and clindamycin
            - immunoglobulin
                - tetanus immunoglobulin should be given IV or IM
                - dose is 5000 to 10,000 international units
            - fractures and dislocations
                - assess and treat accordingly
            - analgesics
                - should be given as required
- common pitfalls
    - history of vaccination does not exclude diagnosis
    - failure to recognise that trismus is characteristic of tetanus
    - failure to perform the spatula test
    - laryngospasm may be severe and occur without warning or may be precipitated by stimulation
    - severe muscular spasms may compromise respiration
    - administration of penicillin when metronidazole is the drug of choice
    - autonomic instability requires active management to reduce the risk of sudden cardiac death
    - neonatal tetanus could present in at-risk groups where maternal immunity is low, for example in patients migrating from areas where vaccine uptake is low
    - the very non-specific presentation may mask the diagnosis

NeuC14 tumours involving the brain and spinal cord

- background
    - preferred terms are high-grade (rapid and aggressive) and low-grade (slow growing) rather than benign or malignant as benign tumours can cause significant morbidity and mortality
- epidemiology
    - primary brain tumours represent around 2% of all tumours diagnosed in the UK
    - lifetime risk of brain or CNS tumour in the UK is 1 in 77
    - incidence of CNS lymphomas is increasing
    - in adults, most brain tumours are supratentorial
        - high grade gliomas and meningiomas predominate
        - most common between 50 and 70 years
- risk factors
    - primary brain tumours are more common amongst affluent group, with the opposite true for brain metastases
    - ionising radiation
    - vinyl chloride is associated with high-grade gliomas
    - immunosuppression (e.g. AIDS) for cerebral lymphoma
    - possible increased risk with:
        - oil refining
        - embalming
        - textile work
    - no definite link with mobile phone use
    - inherited syndromes with increased risk of brain tumour include:

        - neurofibromatosis
        - von Hippel-Lindau disease
        - tuberous sclerosis
        - Li-Fraumeni syndrome
        - Cowden's disease
        - Turcot's syndrome
        - naevoid basal cell carcinoma syndrome (Gorlin's syndrome)
- histological types
    - high grade
        - gliomas and glioblastoma multiforme
        - primary cerebral lymphomas
        - medulloblastomas
    - low grade
        - meningiomas
        - acoustic neuromas
        - neurofibromas
        - pituitary tumours
        - pineal tumours
        - craniopharyngiomas
    - secondaries (10 times more common than primary tumours in adults)
        - common malignancies spreading to brain include:
            - lung cancer
            - breast cancer
            - stomach cancer
            - prostate cancer
            - thyroid cancer
            - colorectal cancer
            - melanoma
            - renal cancer
- presentation
    - depends on location and rate of growth
    - features include:
        - headache
            - typically worse in the mornings
        - nausea and vomiting
        - seizures
        - progressive focal neurological deficit e.g.:
            - diplopia
            - visual field defect
            - upper or lower limb neurological deficit
        - cognitive or behavioural symptoms
        - symptoms relating to location of mass
            - e.g. personality change or disinhibition with frontal lobe lesion
            - dysarthria with parietal lobe lesions
        - papilloedema
- differential
    - other causes of space occupying lesion

    - cerebrovascular event
    - idiopathic intracranial hypertension
- investigations
    - CT
        - may need to progress to contrast scan or MRI
        - may need to image spine with tumours such as germ cell or lymphoma
    - bloods
        - may be useful
        - e.g. for complications of tumour:
            - hypercalcaemia
            - SIADH
        - for other differentials
            - e.g. ESR for possible giant cell arteritis
    - further specialist investigation may include:
        - MRA
        - PET scan
            - useful for grading gliomas or locating occult primary
        - technetium brain scan
            - useful for destructive skull vault or skull base lesions
        - biopsy and tumour removal may be required
- management
    - surgery
        - tumours are resected where possible
            - also provides tissue for formal diagnosis
        - may not be viable if tumour is located in an area associated with critical function or where there is infiltration of local normal brain tissue
        - surgery should be considered to reduce mass effect and treat hydrocephalus for symptomatic relief
        - if surgery is not an option, radiotherapy should be considered
        - photodynamic therapy can be carried out at the same time as surgery – involves injecting a photosensitising agent either intravenously or directly into the tumour then activating in the selected area with a laser (not currently recommended by NICE for routine patient care due to limited evidence of efficacy and safety)
    - radiation
        - external beam radiotherapy can be curative and prolong survival
        - treatment of choice for some types of tumours (e.g. metastatic tumours, leptomeningeal metastases
        - whole brain radiation is used with some tumours (e.g. medulloblastomas, primary CNS lymphomas)
        - in stereotactic radiosurgery, focal radiotherapy is administered to a target, avoiding exposure of normal brain tissue
    - chemotherapy
        - more limited role in brain tumours than in other tumours
            - except for CNS lymphoma, which requires aggressive intrathecal and intravenous chemotherapy

                - important in palliative care and as an adjunct to radiotherapy and surgery
                - commonly used agents include those that can cross the blood brain barrier such as temozolomide in glioblastoma multiforme or platinum agents in medulloblastomas
        - other therapeutic agents
            - may include analgesics, anticonvulsants, anticoagulants, corticosteroids
        - treatment of brain metastases
            - corticosteroids should be used if cerebral oedema is present
            - surgery may be an option if there are 3 or fewer metastases provided the primary is controlled – this is associated with improved survival
            - for metastases 3-3.5cm in size, stereotactic radiosurgery may be an option
            - whole brain radiotherapy can be given after surgery or radiosurgery – there is currently debate over whether it should be given early or late in the illness
                - it is the only treatment modality for those not suitable for surgery or radiosurgery
            - chemotherapy should be considered if the primary is chemosensitive
- complications
    - acute haemorrhage into a tumour
    - blockage of CSF outflow causing hydrocephalus
        - sudden death may result from obstruction of outflow drainage from the third ventricle
    - sudden increases in ICP may lead to life-threatening brain herniation through the foramen magnum or transtentorial foramina
    - complications of radiotherapy
        - acute toxicity is rare but subacute or chronic effects may occur
        - subacute encephalopathy with somnolence and headaches may occur 6-16 weeks after radiation therapy
        - prolonged radiation therapy may lead to impairment of intellectual capacity
- prognosis
    - benign and malignant brain tumours are associated with morbidity relating to mass effect if they increase in size
    - malignant brain tumours are the leading cause of death from solid tumours in children and the third most common cause in adolescents and young adults
- brain tumours in children
    - background
        - most common site for solid tumours in children
        - most (70-80%) are infratentorial tumours (glial tumours, medulloblastoma) or in the midline (germ cell tumours, craniopharyngioma)
        - brain tumours in children generally have a better prognosis than in adults but they are frequently unwell for months prior to diagnosis – prolonged period from symptom onset to diagnosis is associated with poorer prognosis
    - epidemiology
        - brain and CNS tumours are the second most common group of cancers in children and account for a quarter
            - occur in around 5 per 100.000 children aged 0-9 years
        - the largest subgroup is astrocytoma (43%)

        - 76% are low grade and 15% high grade
    - the second most common subgroup is the intracranial and intraspinal embryonal tumours (19%)
- aetiology
    - the underlying cause is unknown
        - some tumours are more common with certain illnesses (e.g. astrocytomas are seen with increased frequency in neurofibromatosis and haemangioblastomas are more prevalent in patients with von Hippel-Lindau disease
    - most cases of brain tumours are sporadic
    - previous cranial irradiation increases the risk of brain tumours (e.g. meningeal leukaemia)
- classification
    - types of brain tumour
        - gliomas (grade 1 is the slowest growing type and grade 4 the fastest growing)
            - astrocytic tumour
                - low grade astrocytoma
                - anaplastic astrocytoma
                - glioblastoma multiforme
            - oligodendroglioma
                - benign
                - anaplastic
            - ependymoma
                - benign
                - anaplastic
            - mixed glioma
                - astrocytoma and oligodendroglioma
            - ganglioglioma
                - benign
                - anaplastic
            - choroid plexus tumour
                - papilloma
                - carcinoma
        - PNETs
            - supratentorial primitive neuroectodermal tumours
            - medulloblastoma
            - pineoblastoma
        - congenital
            - teratoma
            - craniopharyngioma
        - pineal tumours
            - germinoma
            - endodermal sinus tumour
            - embryonal cell carcinoma
            - choriocarcinoma
            - pineocytoma

        - pineoblastoma
    - very rare tumours
        - primary CNS lymphoma
    - benign tumours (more common in adults)
        - meningioma
        - acoustic neuroma
        - pituitary tumour
- presentation
    - intracranial tumours
        - headache
            - usually recurrent, frequent and gradually worsening
            - may be worse on waking (indicating raised ICP)
            - more common with infratentorial lesions
        - nausea and vomiting
        - abnormalities of gait and co-ordination
        - papilloedema
    - children under 4 with intracranial tumours
        - macrocephaly
        - nausea and vomiting
        - irritability
        - lethargy
        - children under 2 tend to have a non-specific presentation with vomiting, lethargy, failure to thrive and irritability
            - they may develop macrocephaly, hyperreflexia and cranial nerve palsies
    - children with intracranial tumour and neurofibromatosis
        - reduced visual acuity
        - exophthalmia
        - optic atrophy
    - posterior fossa tumours
        - nausea and vomiting
        - headache
        - abnormal gait and co-ordination
        - papilloedema
        - presentation of infratentorial tumours relates to blockage of the CSF flow, leading to hydrocephalus
            - common signs and symptoms include morning headache, vomiting, ataxic gait with unsteadiness, double vision, papilloedema
        - brainstem tumours may also present with facial or ocular muscle palsies and hemiparesis
    - supratentorial tumours
        - unspecified symptoms and signs of raised intracranial pressure, seizures and papilloedema
    - central brain tumours
        - headache
        - abnormal eye movements

- squint
- nausea and vomiting

- brainstem tumours
  - abnormal gait and co-ordination
  - cranial nerve palsies
  - pyramidal signs
  - headache
  - squint
- other neurological features related to tumour locations (e.g. personality change in frontal lode tumours, visual defects in occipital lobe tumours)
  - other symptoms relating to type or location of tumour include:
    - chiasmal tumours
      - visual field defects
      - hydrocephalus
    - craniopharyngiomas
      - short stature
      - visual field defects
      - hormonal abnormalities
      - hydrocephalus
    - pineal tumours
      - impaired upgaze
      - impaired accommodation
      - hydrocephalus
    - infratentorial tumours
      - seizures
      - visual problems
      - headaches
      - muscle paralysis
      - respiratory changes
      - increased intracranial pressure
      - poor co-ordination
      - hearing loss
- other features include:
  - weight loss
  - growth failure
  - precocious puberty

- investigations
  - MRI is the preferred form of imaging
    - provides detailed information regarding tumour size, location, extent, surrounding oedema and presence of hydrocephalus
    - may require sedation
    - contrast is usually given
    - CT may used as an alternative
  - biopsy
    - often excision biopsy
  - other investigations depending on presentation
    - may include hearing tests and pituitary function tests

- management
  - depends on tumour type and location and the age of the child
  - surgical resection
    - complete total resection should always be the aim and is associated with improved survival
      - rarely achievable as the margins of most tumours are indistinct and it is difficult to determine whether tissue is normal or abnormal
    - biopsy may be performed beforehand – usually direct open biopsy at the time of surgery, but stereotactic biopsies are taken for some tumours such as for basal ganglia and brainstem lesions
      - children may be given phenytoin and corticosteroids preoperatively
    - hydrocephalus is common postoperatively and an external ventricular drain or ventriculoperitoneal shunt is inserted for removal a few days later when the CSF clears
    - very young children (under 2) require radical resection as radiotherapy is delayed until they are older as it will damage developing tissue; usually followed by chemotherapy
  - radiotherapy
    - provided in very low doses to very localised areas to avoid damage to normal surrounding brain tissue
    - various techniques such as gamma knife therapy (used for slow-growing lesions) and interstitial seeds
  - chemotherapy
    - various regimes, usually involving vincristine
    - has good outcomes used alone for rare primary CNS lymphoma
    - used for low grade gliomas if there is residual disease after resection
- follow up
  - children have MRI scans every six months for the first two years, then annually
- complications
  - intellectual decline
    - likely multifactorial – local tumour effects, surgery and radiotherapy
  - growth hormone deficiency – common
  - neurological handicap – may be permanent
  - increased risk of second brain tumour 10-20 years later due to irradiation (e.g. meningioma or sarcoma) – increased risk if brain irradiated at a very young age
  - reduced bone density – multifactorial
  - cavernomas presenting as haemorrhagic lesions are associated with CNS irradiation
- prognosis
  - earlier diagnosis improves long-term outcomes
  - resection of the tumour may resolve seizures and headaches
  - surgical mortality is 1% for a paediatric craniotomy
    - morbidity is higher and depends on pre-operative condition

        - tumours of the brain and CNS are the most common cause of deaths from cancer in childhood
- spinal tumours
    - background
        - primary non-proliferative tumours of the spine are uncommon
            - less than 5% of bone neoplasms
        - metastatic disease of the spine is much more common
            - 40-80% of patients who die from cancer have bony metastases at time of death and the spine is the most common location
        - problems can be caused by direct compression of the spinal cord, vascular obstruction and invasive infiltration
    - primary spinal tumours
        - bone-producing tumours of the spine
            - osteoid osteoma
                - benign and locally self-limiting
                - typically in children aged 10-20 years, mostly males
                - involve axial skeleton 10% of the time
                - over half are found in the lumbar region
                    - next most common site is cervical, then thoracic, then sacral
                - usually symptomatic
                - can result in:
                    - painful scoliosis
                    - muscular atrophy
                    - radicular pain
                    - gait disturbances secondary to pain and splinting
            - osteoblastoma
                - benign but expand locally and are aggressive
                - occur in second or third decade of life
                - twice as common in males
                - typically cause dull localised pain and paraesthesiae
                - may cause paraparesis, and, if large enough, paralysis
            - giant cell tumours
                - most are benign
                - malignancy occurs in a minority and is usually related to previous irradiation in the area of the tumour
                - more common in women and in the third to fifth decades of life
                - can increase dramatically in size during pregnancy secondary to hormonal influences
                - symptoms include pain with radicular pattern and neurological impingement with weakness and sensory deficits
            - osteosarcoma
                - malignant spindle cell lesion which produces osteoid, the organic portion of the bone matrix, secreted by osteoblasts
                - rare in the spine

        - typically present in the fourth decade of life
        - male predominance
        - most common lumbosacrally
        - often present with pain and a palpable mass
        - neurological symptoms are found in the majority of patients
        - serum alkaline phosphatase may be elevated
- cartilage-producing tumours of the spine
    - osteochondroma
        - commonly referred to as exostosis
        - benign lesion with cartilaginous cap
        - 4% of all solitary spine tumours
        - occur in patients aged 20-30
        - more common in males
        - more common in the cervical spine
    - chondrosarcoma
        - malignant cartilage-producing tumour
        - second most common non-lymphoproliferative tumour of the spine
        - 10% of all spinal tumours
        - more common in men
        - mean age at presentation is 45 years
        - thoracic spine is most common site
        - most common symptoms are pain, palpable mass and neurological complaints
- lymphoproliferative tumours
    - multiple myeloma and plasmacytoma
        - multiple myeloma is the most common primary malignancy of bone and spine
        - systemic disease affecting middle aged people and characterised by areas of local bone destruction
        - underlying cell is the malignant plasma cell, producing abnormal quantities of immunoglobulins
        - patients complain of pain that may be worse at night
    - lymphoma
        - associated with a large infiltrate of lymphoid cells
- tumour of notochordal origin
    - chordoma
        - uncommon but are the most common primary malignant tumour of the spine in the adult
        - presentation is often subtle with gradual onset of pain, numbness, motor weakness and constipation or incontinence
        - constipation is a uniform finding in most patients with sacrococcygeal lesions
        - slow growing lesions and are often very large at the time of presentation
- round cell tumour

        - Ewing's sarcoma
            - most common non-lymphoproliferative primary malignant tumour of the spine in children
            - usually presents at 10-20 years
            - most common site in the spine is sacrococcygeal region
    - metastatic tumours
        - most common tumours metastasising to the spine are:
            - prostate cancer
            - breast cancer
            - lung cancer
            - renal cancer
            - gastric cancer
- presentation
    - symptoms
        - systemic or constitutional symptoms tend to be more common with malignant or metastatic disease than in benign lesions
        - symptoms usually develop slowly
        - back pain is the most frequent symptom
            - with extradural lesions pain is typically aggravated by coughing or straining
            - it may be radicular, localised to the back or felt generally in an extremity
        - neurological deficits secondary to compression of the spinal cord or nerve roots can be part of the presentation
            - degree of neurological compromise can vary from slight weakness or an abnormal reflex to complete paraplegia, depending on degree of encroachment
            - loss of bowel or bladder continence can occur from neurological compression or secondary to mass effect
            - there may be motor deficits, paraesthesia, numbness in legs and loss of sphincter control
    - signs
        - local spinal tenderness
        - motor deficits
        - sensory changes
- investigations
    - bloods
        - FBC for WCC and Hb
        - U&Es
        - ESR or CRP may be useful
        - calcium and ALP may provide evidence of neoplastic bone process
        - serum electrophoresis may be indicated
    - imaging
        - x-ray, CT or MRI
    - confirmation of diagnosis and tumour type is by biopsy of the spine lesion after imaging – may be open or percutaneous image-guided
- management

- decompression and excision where possible, radiotherapy and chemotherapy where indicated
- osteoid osteomas
    - symptoms respond to NSAIDs or salicylates
    - treatment is resection of the nidus by open surgical approach or percutaneous CT guided resection
- osteoblastomas
    - wide local resection where possible
        - sometimes limited by proximity of vital vessels or neural tissue
    - tumour recurrence may occur
- giant cell tumour
    - lesions are locally aggressive and size and location may not allow complete resection
    - those that cannot be excised should be curetted
    - radiation is reserved for surgically inaccessible tumours
    - recurrence rates can be up to 50%
- osteochondroma
    - complete surgical resection is usually curative
    - clinical symptoms usually improve following removal of the exostosis
    - incomplete resection can lead to recurrence
- chondrosarcoma
    - complete resection of the vertebra and strut bone grafting may be necessary for complete excision
    - cure is possible when complete resection can be achieved but this is only possible in a minority of cases
    - if wide marginal resection cannot be achieved, the tumour recurrence and mortality rates are high
    - chemotherapy is sometimes used to reduce the size of the mass
    - metastases depend on the grade of the primary chondrosarcoma – the lungs are the most frequent site
- Ewing's sarcoma
    - radiation and chemotherapy are the mainstays of treatment for Ewing's sarcoma in the spine
    - there is almost 100% local control and excellent long-term survival in patients with non-sacral sarcomas
    - sacral tumours have a lower control rate and much poorer long-term survival due to the tendency for delayed presentation and larger size
    - most important prognostic indicator for survival is response to chemotherapy
- osteosarcoma
    - surgical resection is the treatment of choice but often incomplete due to size and location
    - adjuvant chemotherapy and radiotherapy are often used with varying degrees of success

                - spinal osteosarcomas have a very poor prognosis with death usually occurring within the first year after diagnosis
                - only a few patients have survived longer than two years
            - chordomas
                - surgical resection is the treatment of choice
                - prognosis depends on whether the tumour can be resected completely
                - patients with sacrococcygeal tumours often have improved survival because the surrounding structures are more expendable and allow better resection
                    - these patients usually have 8-10 years of survival as opposed to 4-5 years for other spinal sites
                - death is usually related to local recurrence and invasion rather than metastatic disease
                    - if they do metastasis, the most common sites are liver, lungs, regional lymph nodes, peritoneum, skin and heart
            - multiple myeloma
                - generally sensitive to radiation and chemotherapy
                - surgery for stabilisation is indicated in myelomas of the spine when there is extensive destruction of the vertebral body with collapse and possible kyphosis with canal compromise
                    - adjuvant radiation therapy may be used postoperatively once the surgical site has healed
    - complications
        - complications associated with the tumour, its recurrence or metastases
            - neurological complications including radicular pain or focal weakness from impingement on a nerve root
            - complete or incomplete paraplegia from direct pressure on the spinal cord
        - complications associated with the surgical, radiation or chemotherapeutic treatment
            - e.g. related to structures sacrificed during surgical resection, structures in the path of the radiation therapy or systemic effects of chemotherapy

NeuC15 VP shunts

- background
    - a medical device used to drain fluid via a pressure gradient away from the brain for conditions with excessive cerebrospinal fluid
        - shunting fluid away avoids excessive pressure on the brain
    - shunts drain according to the differential pressure gradient between the ventricle and the tip of the distal catheter
    - the ventricular end of the catheter is inserted through a burr hole in the right parieto-occipital region and the valve often sits behind the right ear
    - the distal portion is subcutaneously tunnelled down into the abdomen where it is positioned inside the peritoneal cavity
    - there are three shunt types:

        - ventriculoperitoneal (VP) – the most common
        - ventriculopleural (VPL)
        - ventriculoatrial (VA)
- pathophysiology
    - CSF is produced at a rate of around 20ml/h in children and adults
        - in a normal adult it is recycled three times a day
    - normal adult volumes of CSF (around 150ml) are reached by age 5
        - the volume in a neonate can be estimated at 2ml/kg
    - around 50% of CSF is created by the choroid plexus of the lateral, third and fourth ventricles
        - the rest of the CSF arises from the extracellular fluid of the brain
    - CSF travels out of the foramen of Lushka and foramen of Magendie (at the level of the fourth ventricle) through to the subarachnoid spaces, along the spinal cord to the cerebral hemispheres
        - it is reabsorbed by arachnoid villi and moves into the venous sinuses which drain into the internal jugular veins
    - intracranial pressure rises when CSF production exceeds absorption; the consequence is hydrocephalus
        - it can be caused by disruptions in formation, flow or absorption
        - as hydrocephalus worsens and intracranial pressure increases, the temporal and frontal horns dilate, sometimes asymmetrically, with elevation of the corpus callosum and stretching of white matter tracts
- indications
    - congenital
        - spina bifida (15-20%)
        - congenital hydrocephalus (13-22%)
        - chiari malformation (type 1 or 2) (1-2%)
        - aqueductal stenosis (2-5%)
        - dandy-walker syndrome (1-5%)
        - craniosynostosis (1%)
    - acquired
        - tumour (10%)
        - intraventricular haemorrhage (3-15%)
        - infectious (3-4%)
        - arachnoid cyst (3%)
        - trauma (1-2%)
- concerns for patients and parents
    - flying
        - no evidence that this is dangerous
        - patients can carry shunt alert cards displaying the type of shunt
        - they also require augmented travel insurance
    - sports
        - contact sports such as boxing are contraindicated
        - in the UK neurosurgeons advise that football and rugby are acceptable wearing a skull cap
        - patients can go scuba diving
    - employment

        - in the UK, the Royal Air Force, Royal Navy and police force do not allow shunts
    - programmable shunts and magnets
        - background magnetic fields of household appliances are safe, as are metal detectors
        - all are MRI safe but post MRI check is recommended with some
        - the iPad2 has a strong magnetic field and can reprogramme some shunt valves so should be kept at safe distances
    - shunt length
        - reassurance that placing sufficient length inside the abdomen will suffice and allow for growth
    - weight
        - obesity is a risk factor for failure of VP shunts and dislodgement
- shunt-related complications
    - up to 50% failure rate by two years with median time to shunt failure 1.5 years
    - paediatric revisions are more common than adult revisions
    - reasons for failure include:
        - proximal occlusion
        - distal occlusion
        - disconnection
        - distal disconnection/migration
        - infection (acute and late)
        - proximal displacement
        - distal displacement
        - valve defect
        - over drainage
- risk factors for shunt failure include:
    - younger patients (<6 months), particularly neonates
    - complex comorbidities
        - e.g. cardiac, myelomeningocele, tumour, congenital hydrocephalus
    - prior shunt failure and short time intervals between revisions
    - male sex
    - low socioeconomic status
- causes of shunt failure
    - obstruction, blockage or occlusion
        - the commonest cause is proximal occlusion
        - it can be occluded by brain parenchyma on insertion, blood following choroid plexus haemorrhage, other cellular matter (e.g. macrophages, giant cells, connective tissue, fibrin networks, debris, neoplastic cells)
        - distal blockage tends to occur later and should raise suspicion for intra-abdominal pseudocyst or adhesions, which may affect future peritoneal catheter placements
    - shunt infection
        - second most common reason for malfunction
        - risk factors include:
            - young age
            - post-op CSF leak

            - previous shunt infection
            - presence of gastrostomy tube
        - the majority of infections are acute, during the first 6 months
        - late onset infections are mostly attributable to peritonitis, abdominal pseudocyst, bowel perforation or haematogenous inoculation
        - shunt infection is associated with an increased risk of seizure disorder, decreased intellectual performance and a two-fold increase in long-term mortality
        - re-infection occurs in a quarter
        - fever is common
        - the presence of >10% neutrophils in a sample of CSF from the shunt is highly specific and sensitive for infection
        - commonest organisms are skin flora including Staph. epidermidis, Staph. aureus and Gram-negative rods
    - shunt fracture
        - often a late complication
        - almost always occurs along the distal portion between the valve and peritoneum
        - with age, fibrous tissue becomes calcified and does not slide freely within the subcutaneous tissue, which can cause the tubing to crack
            - it is more likely to happen in the neck where more movement occurs
        - tension can also form along areas of calcification causing tethering and stretching as a child grows
        - early shunt fracture can occur and can be a consequence of trauma to the tubing during surgery
        - shunt series x-rays should be done
    - shunt disconnections
        - the catheters are generally multi-component and hubbed together, so disconnections can occur soon after surgery
        - the disconnection impedes the flow of CSF and it may still leak
        - onset of symptoms may be slow
        - disconnections can happen at either the proximal or distal aspect of the valve
    - abdominal pseudocyst
        - a rare complication of VP shunts
        - usually a late complication occurring years after initial placement
        - it is a fluid filled sac that collects at the distal end of the catheter, possibly due to inflammation or abdominal adhesions
        - can present with abdominal pain or distension with or without a palpable mass
        - neurological symptoms occur when there is elevated ICP
    - shunt migration
        - the proximal or distal catheter tip may migrate
        - rarely, the proximal catheter can withdraw from the ventricle, or the distal catheter can shift away from the peritoneum
        - the distal tubing can become tethered and cause traction on some of the components causing a disconnection

        - distal migration occurs as a child grows
    - over-drainage
        - with rapid over-drainage, the dura can be stressed and subdural haematomas and/or extra-axial fluid collections can form
- clinical presentation of shunt malfunction
    - may include:
        - headache
        - nausea
        - vomiting
        - fever
        - irritability
        - abnormal level of consciousness
    - infants:
        - difficulty feeding
        - bulging fontanelle
    - older children:
        - nausea
        - somnolence
        - lethargy
        - cognitive difficulties
        - eye pain
    - adults:
        - headache
        - nausea and vomiting
        - lethargy
        - ataxia
        - altered mental status
        - paralysis of upward gaze ('sunset eyes')
        - dilated pupils
        - cranial nerve palsies
    - those with shunts because of myelomeningoceles may present with:
        - weakness
        - difficulty walking
        - bowel/bladder dysfunction
        - lower cranial nerve palsies
- examination
    - unlikely to find anything specific
    - may show:
        - decreased level of consciousness
        - erythema along the shunt tract
        - bulging fontanelle
        - nausea/vomiting
        - irritability
    - valve chamber abnormality
        - gently compress the chamber and observe for refill
        - difficulty compressing the chamber indicates distal flow obstruction
        - slow refill (>3s) indicates proximal obstruction
- diagnosis

    - CT
        - likely to show an increase in ventricular size
        - sometimes shows periventricular lucency representing oedema
        - 15% of patients do not have ventricles that enlarge with shunt failure and raised ICP
    - lumbar puncture
        - may demonstrate increased opening pressures (not always)
        - also used for evidence of infection
        - not commonly performed in the ED
    - shunt series
        - used to check the overall course of the catheter looking for disconnection or disruption
        - it will not show obstruction, only damage to the catheter
        - it can rarely demonstrate complications such as CSF pseudocyst but cannot be relied on for this
        - usually requires 3-4 images (depending on the size of the child), including two views of the skull and the continuous trajectory of the shunt tubing down the neck, chest and looping into the abdomen
- management
    - low threshold for contacting neurosurgery for advice

NeuC16 Wernicke's encephalopathy

- background
    - Wernicke-Korsakoff syndrome is a spectrum of disease resulting from thiamine deficiency
    - Korsakoff's is the late manifestation of the syndrome
    - alcohol-related brain damage may contribute to up to a quarter of cases of dementia
    - incidence of Korsakoff's is rising
        - prevalence is higher in the homeless, people living alone or in isolation and psychiatric inpatients
- pathogenesis
    - chronic alcohol consumption can result in thiamine deficiency by causing:
        - inadequate nutritional thiamine intake
        - decreased absorption of thiamine from the gastrointestinal tract
        - impaired thiamine utilisation in the cells
    - susceptibility differs and different brain regions may be more or less sensitive
    - thiamine is a cofactor required by three enzymes in pathways of carbohydrate metabolism
        - a reduction in thiamine can interfere with numerous cellular functions
        - alcohol related neuronal loss mainly affects the thalamus, hypothalamus and mamillary bodies
    - chronic subdural haematoma has been documented as a cause of Wernicke's, with symptoms persisting after resolution due to organic atrophic changes of the frontal and temporal lobes due to long term compression
    - it has also been reported following nutritional stress such as laparotomy for small bowel obstruction, bariatric surgery or rapid weight loss
    - other chronic conditions that may cause thiamine deficiency include:

        - AIDS
        - hyperemesis gravidarum
        - thyrotoxicosis
        - cancers with metastasis
        - long term dialysis
        - congestive heart failure treated with long term diuretic therapy
- presentation
    - any patient with alcohol misuse presenting with confusion, nausea, vomiting, fatigue, weakness or apathy, or with unexplained hypotension or hypothermia should be considered at risk
    - symptoms
        - vision changes
            - double vision
            - eye movement abnormalities
            - eyelid drooping
        - loss of muscle co-ordination
            - unsteady, unco-ordinated walking
        - loss of memory, which can be profound
        - inability to form new memories
        - hallucinations
    - signs
        - the patient is usually alert, with vocabulary, comprehension, motor skills, social habits and naming ability maintained
        - CNS examination may show polyneuropathy
        - reflexes may be decreased or abnormal
        - gait and co-ordination are abnormal
        - muscles may be weak and show atrophy
        - eyes show abnormality of movement
            - nystagmus
            - bilateral lateral rectus palsy
            - conjugate gaze palsy
        - blood pressure and body temperature may be low
        - pulse may be rapid
        - the person may appear cachectic
    - cognitive features
        - confabulation
            - falsification of memory in clear consciousness
            - answering questions promptly with inaccurate and sometimes bizarre answers
        - memory loss
            - anterograde amnesia is the main feature – loss of memory for events occurring after the onset of the disorder
            - inability to learn and repeat simple pieces of information or learn new tasks
            - often disoriented in time and place
        - retrograde amnesia
            - loss of memory for events before onset of the disorder

        - some memory of distant events may be preserved
        - telescoping of event is common – patient says something happened recently when it took place years ago
- diagnosis of encephalopathy
    - at least two of:
        - dietary deficiencies
        - oculomotor abnormalities
        - cerebellar dysfunction
        - altered mental state or mild memory impairment
    - subclinical episodes can occur
- differential
    - drug misuse
    - alcoholic ketoacidosis
    - delirium tremens
    - chronic hypoxia
    - head injury
    - hepatic encephalopathy
    - post-ictal state
    - cerebrovascular disease
    - brain tumour
- investigations
    - diagnosis is based mainly on history and examination
    - bloods:
        - FBC – raised MCV
        - U&Es/LFTs/bone profile – hypernatraemia, hypercalcaemia, uraemia
        - glucose
        - consider ABG to rule out hypercarbia/hypoxia
        - cholesterol
        - serum thiamine (B1) levels
    - LP may be required to exclude nonfocal CNS infection
    - imaging
        - CT head may be considered
        - MRI
    - EEG – may be considered to rule out non-convulsive status epilepticus
- associated diseases
    - peripheral neuropathy
    - paraesthesia
    - malnutrition
    - liver disease
    - delirium tremens (around 10%)
    - beriberi (around 5%)
- management
    - Pabrinex IV 2-3 pairs over 30 minutes
        - IV pair contains 250mg thiamine, 4mg riboflavin and 50mg pyridoxine (first ampoule) and 500mg ascorbic acid, 160mg nicotinamide and 1000mg glucose (second ampoule)
        - IM pair is the same without the glucose

        - three times a day for three days
        - can rarely cause serious allergic adverse reactions
        - there is no evidence that treating hypoglycaemia before giving thiamine precipitates Wernicke's
    - oral treatment
        - thiamine and vitamin B complex in the long term
        - harmful or dependent drinkers should be offered thiamine if they are malnourished (50mg OD) or if they have decompensated liver disease
        - the BNF recommends 25-100mg/day for mild thiamine deficiency and 200-300mg/day for severe deficiency
    - ongoing management for those with persistent symptoms
        - multidisciplinary team approach
        - management of alcohol misuse
        - assessment of memory impairment
        - home support or residential care may be required, with occupational therapy input
        - some patients may require consideration of the Mental Capacity Act or sometimes the Mental Health Act
- complications
    - development of Korsakoff's syndrome
        - can develop in untreated or undertreated Wernicke's
    - symptoms of alcohol withdrawal (tremors, hallucinations, convulsions) can complicate the picture
    - recurrences of encephalopathy can occur in patients who continue to drink and fail to maintain their thiamine intake
    - congestive heart failure can occur
    - fine horizontal nystagmus can persist, although vertical nystagmus may slowly resolve and most other ocular symptoms rapidly resolve
    - ataxia may persist and cause a slow shuffling gait
    - global confusion may be slow to resolve and there may be persisting learning difficulties and memory impairment
- prognosis
    - untreated, Wernicke's encephalopathy leads to death in up to 20% and to Korsakoff's in 85% of survivors
    - up to 25% of patients with Korsakoff's require long-term institutionalisation
- prevention
    - alcohol avoidance
    - thiamine supplementation for patients with significant alcohol dependency
    - considering homeless patients with inadequate social support to be at greatest risk

OBSTETRICS AND GYNAECOLOGY

ObP1 pelvic pain

- acute pelvic pain
    - background
        - more common in women

        - most women have mild pelvic pain at some point due to periods, ovulation or sexual intercourse
        - when severe, it is the most common indication for laparoscopy
    - aetiology
        - common causes:
            - pelvic inflammatory disease
            - urinary tract infection
            - miscarriage
            - ectopic pregnancy
            - torsion or rupture of ovarian cyst
        - pregnancy related:
            - miscarriage
            - ectopic pregnancy
            - rupture of corpeus luteum cyst
            - premature labour
            - placental abruption
            - uterine rupture
        - gynaecological:
            - ovulation
            - dysmenorrhoea
            - PID
            - rupture or torsion of ovarian cyst
            - degenerative changes in a fibroid
            - pelvic tumour
            - pelvic vein thrombosis
        - other:
            - appendicitis
            - irritable bowel syndrome
            - adhesions
            - prostatitis
            - strangulated hernia
    - investigations
        - may include:
            - urinalysis, MSU
            - high vaginal swab and endocervical swab
            - pregnancy test
            - FBC
            - urgent ultrasound if miscarriage/ectopic suspected
            - laparoscopy
- chronic pelvic pain
    - background
        - much more common in women
        - may occur in as many as 1 in 6 adult women
        - defined as:
            - intermittent or constant pain in the lower abdomen (or pelvis in women)

- lasting for at least six months
- not occurring exclusively with menstruation or sexual intercourse
- not associated with pregnancy

- aetiology
  - not well understood
  - often has more than one cause
  - pain may persist long after the original tissue injury has healed
  - psychological, social and physical factors are all important
  - persistence of pain may lead to changes in the central nervous system which magnify the original signal
  - possible causes of chronic pelvic pain include:
    - endometriosis
      - pain usually varies during menstrual cycle
      - can be associated with dysmenorrhoea and dyspareunia
    - adhesions
      - may be caused by previous surgery, endometriosis, previous infection
      - some adhesions are asymptomatic
    - IBS
    - interstitial cystitis
    - musculoskeletal problems
    - pelvic organ prolapse
    - nerve entrapment
      - can occur in scar tissue or fascia
    - psychological and social issues
      - depression and sleep disorders are common
      - women with chronic pelvic pain are more likely to have experienced physical or sexual abuse as children
    - other causes in men include epididymo-orchitis and testicular tumours
    - chronic pain in the region of the prostate is referred to as prostate pain syndrome
- presentation
  - initial history about the pattern of the pain and its association with other symptoms, including bladder and bowel symptoms and the effect of movement and posture on the pain
  - psychological and social issues
  - variation with the menstrual cycle
  - red flag symptoms and signs:
    - bleeding per rectum
    - new bowel symptoms in patients over 50 years old
    - new pain after the menopause
    - pelvic mass
    - suicidal ideation
    - excessive weight loss
    - irregular vaginal bleeding in patients over 40
    - postcoital bleeding

- investigations
    - may include:
        - samples to screen for infection in those who are sexually active
        - bloods such as FBC, CRP
        - CA125 if symptoms suggestive of ovarian cancer (including symptoms suggestive of irritable bowel syndrome in women over 50
        - urinalysis and MSU
        - transvaginal ultrasound to screen for and assess adnexal masses
        - TVS and MRI for adenomysosis
        - consideration of laparoscopy if other therapeutic interventions fail
        - further urological or bowel investigations
- management
    - focused on identifying and treating the cause
    - needs to include psychosocial causes and effects
    - a symptom diary may be useful
    - women with cyclical pain should consider a trial of the oral contraceptive pill or GnRH agonist for three to six months before diagnostic laparoscopy
    - analgesia +/- referral to pain clinic
    - for men with PPS:
        - alpha blockers for those with symptoms for <1 year
        - antibiotics (quinolones or tetracyclines) for at least 6 weeks for those with symptoms for <1 year
        - NSAIDs may be effective
        - consideration of electro-acupuncture or perineal extra-corporeal shock wave therapy

ObP2 vaginal bleeding

- intermenstrual and postcoital bleeding
    - background
        - intermenstrual bleeding is vaginal bleeding (other than postcoital) at any time during the menstrual cycle other than during normal menstruation
        - postcoital bleeding is non-menstrual bleeding that occurs immediately after sexual intercourse
        - both are symptoms rather than diagnoses, and require further investigation
        - around 14% of pre-menopausal women have irregular or excessively heavy menstrual bleeding
    - aetiology
        - postcoital bleeding
            - infection
            - cervical ectropion
                - especially in women taking the combined oral contraceptive pill
            - cervical or endometrial polyps
            - vaginal cancer
            - cervical cancer
                - usually apparent on speculum examination
            - trauma or sexual abuse

- vaginal atrophic change
- no specific cause found in around 50%

- intermenstrual bleeding
  - pregnancy-related
    - including ectopic pregnancy and gestational trophoblastic disease
  - physiological
    - vaginal spotting around ovulation
    - hormonal fluctuation during perimenopause
      - should be a diagnosis of exclusion
  - vaginal causes
    - adenosis
    - vaginitis
      - bleeding uncommon before menopause
    - tumours
  - cervical causes
    - infection
      - chlamydia
      - gonorrhoea
    - cancer
      - bleeding is most often postcoital
    - cervical polyps
    - cervical ectropion
    - condylomata acuminata of the cervix
  - uterine causes
    - fibroids
      - occur in 25% of women of reproductive age
    - endometrial polyps
    - cancer
      - endometrial adenocarcinoma
      - adenosarcoma
      - leiomyosarcoma
    - adenomyosis
      - usually only symptomatic in later reproductive years
    - endometritis
  - oestrogen-secreting ovarian cancers
  - iatrogenic causes
    - tamoxifen
    - following smear or treatment to the cervix
    - missed oral contraceptive pills
    - drugs altering clotting parameters
      - including anticoagulants, SSRIs, steroids
    - alternative remedies when taken with hormonal contraceptives
      - e.g. ginseng, gingko, soy supplements, St John's wort

- presentation

- history
    - menstrual history
        - last menstrual period and whether it was ‘normal’
        - regularity and cycle length
        - duration of abnormal bleeding (prolonged or recent)
        - presence of menorrhagia
        - timing of bleeding in the menstrual cycle
        - associated symptoms
            - abdominal pain
            - fever
            - vaginal discharge
            - dyspareunia
        - factors that aggravate bleeding
            - e.g. exercise, intercourse
    - obstetric history
        - previous pregnancies and deliveries, including time since last delivery/miscarriage/termination
        - current breastfeeding
        - risk of current pregnancy
            - e.g. unprotected intercourse, forgotten pills, gastroenteritis
        - risk factors for ectopic pregnancy
            - history of PID
            - history of endometriosis
            - IVF treatment
            - intrauterine contraceptive device
            - POP
    - gynaecological history
        - current use of contraception
        - smears
            - most recent results
            - any previous abnormalities
            - colposcopy
            - treatment for abnormalities
    - sexual history
        - risk factors for STI
        - past history of and treatment for STIs
    - medical history
        - e.g. bleeding disorders, diabetes
    - current medication, including unprescribed
- examination
    - establish that bleeding is from the vagina, not the rectum or in the urine
        - can ask the patient to insert a tampon to confirm
    - BMI
        - high BMI is an independent risk factor for endometrial cancer

- abdominal examination – presence or absence of pelvic masses
- PV examination (speculum and bimanual)
    - contact bleeding
    - friability of tissue
    - cervical excitation or tenderness
    - ulceration, polyps or discharge
    - cervical ectropion
        - red ring around the external os due to extension of the endocervical columnar epithelium over the ectocervix
    - cervical polyp
        - mass arising from the endocervix, usually protruding from the external os into the vagina
        - can be avulsed and sent for histology
        - endometrial polyps can occasionally be seen extruding though the cervix
    - cervicitis
        - cervix appears red, congested and sometimes oedematous
        - may be purulent discharge with the cervix tender to palpation
        - most common cause is chlamydia

- investigations
    - pregnancy test
    - infection screen
    - blood tests may include:
        - FBC
        - clotting
        - TFTs
        - FSH/LH levels if menopause suspected
    - transvaginal ultrasound
        - investigation of choice to look for structural abnormality
        - ultrasound should be done immediately postmenstrually when the endometrium is thinnest and polyps and cystic areas are more obvious
    - endometrial biopsy
    - colposcopy
- red flags for urgent referral include:
    - abnormal-looking cervix suspicious of cervical cancer
    - cervical polyp not easily removable of suspicious looking
    - pelvic mass on examination or significant abnormality on US
    - women at high risk of endometrial cancer:
        - family history of hormone-dependent cancer
        - prolonged and irregular cycles
        - taking tamoxifen
    - over 45 with intermenstrual bleeding or under 45 with persistent symptoms or risk factors for endometrial cancer

        - non-urgent referral for women with no cause found on examination for postcoital bleeding
    - management
        - for patients on the contraceptive pill, generally waiting 3 months after starting to see if symptoms subside before considering alternatives
            - pregnancy test must be done
        - cervical ectropions
            - may resolve spontaneously if COC pill is stopped or following pregnancy
            - can be treated conservatively or cauterised with silver nitrate
            - other treatment options include thermal cautery and diathermy, cryosurgery, laser or microwave therapy
        - cervical and endometrial polyps
            - cervical polyps should be sent for histology
        - fibroids
            - small fibroids can be removed hysteroscopically
            - uterine artery embolization can be effective
            - medical management includes drugs that reduce oestrogen levels
            - women with larger fibroids can be treated with drugs, vascular embolization, surgery or a combination
- postmenopausal bleeding
    - background
        - vaginal bleeding occurring after twelve months of amenorrhoea in a woman of the age where the menopause can be expected
        - usually has a benign cause, but the priority is to exclude malignancy
        - common problem, representing 5% of all gynaecology outpatient attendances
    - risk factors for endometrial cancer
        - unopposed oestrogen-only HRT
        - tamoxifen use
            - anti-oestrogen effect on breast, pro oestrogen effect on uterus and bones
        - low parity or infertility
        - early menarche and late menopause
        - increasing age
        - polycystic ovary disease
        - hereditary non-polyposis colorectal carcinoma
        - obesity combined with diabetes
        - hypertension
    - protective factors for endometrial cancer
        - COC pill
        - grand multiparity
    - postmenopausal bleeding aetiology
        - vaginal atrophy
            - most common cause
        - use of HRT
        - endometrial hyperplasia

        - endometrial cancer
        - endometrial polyps or cervical polyps
        - cervical cancer
        - uterine sarcoma (rare)
        - ovarian cancer
        - vaginal cancer (very uncommon)
        - non-gynaecological causes including trauma or a bleeding disorder
    - management
        - should be treated as malignant until proven otherwise with two week wait referral
    - investigations include
        - transvaginal US
            - the endometrium is postmenopausal women is thinner, and thickening may indicate pathology
        - endometrial biopsy
        - hysteroscopy
- vaginal cancer
    - background
        - usually a squamous cell carcinoma of the posterior wall of the upper third of the vagina
        - may directly invade bladder or rectum
        - metastasis is to regional lymph nodes and para-aortic nodes
        - around 10% are adenocarcinoma which has a peak incidence between 17 and 21 years and has an increase in pulmonary metastases and supraclavicular/pelvic node involvement
        - 80% of vaginal cancer is metastatic spread, such as from the urethra, bladder, Bartholin's gland, rectum, endometrium, kidney, ovary or endocervix
        - rare, accounting for 1% of all gynaecological cancers
    - presentation
        - vaginal bleeding or bloody discharge
        - pain or leg oedema in advanced tumours
    - investigations
        - colposcopy
        - biopsy, cervical cytology, endometrial biopsy
        - CT scan/PET scan
        - CXR
        - cystoscopy, sigmoidoscopy
    - staging (International Federation of Gynaecology and Obstetrics)
        - stage 0 – squamous cell carcinoma in situ
        - stage I – disease limited to the vaginal wall mucosa
        - stage 2 – disease involved the subvaginal tissue but not the pelvic wall
        - stage III – disease extends to the pelvic wall
        - stage IV – disease either extends beyond the true pelvis or involves the bladder or rectal mucosa
            - stage Iva – disease has spread to adjacent organs
            - stage IVb – disease has spread to distant organs
    - management

        - surgery and radiotherapy are effective in early-stage disease
        - radiation therapy is the primary treatment for more advanced stages
    - prognosis
        - depends on stage
        - survival reduced in patients who:
            - are older than 60
            - are symptomatic at time of diagnosis
            - have lesions of the middle and lower third of the vagina
            - have adenocarcinoma
            - have poorly differentiated tumours

ObP3 pregnancy

- changes in pregnancy
    - breast
        - mammogenesis occurs in early pregnancy
            - breasts enlarge via cellular hyperplasia and the breast lobules increase in size
            - nipple become erectile, the areola becomes larger and darker and superficial veins can become visible
            - breasts may be tender or painful
        - lactogenesis begins towards the middle of pregnancy
            - epithelial cells change into secretory epithelium
            - towards the end, alveoli secrete colostrum
            - after birth, the influence of progesterone abruptly ceases and the onset of milk production begins
    - uterus
        - grows at a steady, predictable rate
        - shape changes from the nonpregnant pear shape to a sphere, then an elongated cylinder
        - the round ligaments hypertrophy and stretch
        - once a blastocyst implants, there is transformation of the endometrium into the decidua
    - cervical remodelling
        - glandular tissue produces thick, tenacious mucus which forms the plug sealing the endocervical canal
        - the cervix has four distinct phases:
            - softening/remodelling
            - accelerated softening at the end of pregnancy
            - dilation
            - repair
- hormones and their function
    - human chorionic gonadotrophin
        - source
            - syncytiotrophoblast
            - placenta
        - functions
            - stimulates production of progesterone from the corpus luteum

                - prevents degeneration of the corpus luteum
                - promotes formation of the syncytiotrophoblast during trophoblastic invasion
                - aids spiral artery remodelling
                - promotes angiogenesis in the uterine vasculature
                - stimulates thyroid production of thyroxine in first trimester
                - suppresses myometrial contractions
        - human placental lactogen
            - source
                - placenta
            - functions
                - increases insulin resistance
                - stimulate production of growth hormones
        - progesterone
            - source
                - corpus luteum
                - placenta
            - functions
                - promotes systemic vasodilation
                - anti-inflammatory properties protect trophoblast from being rejected
                - supports decidualisation
                - prevents myometrial contractility
                - inhibits uterine production of prostaglandins
                - supports mammary growth for lactation
                - withdrawal at term leads to uterine contractions
        - oestrogen
            - source
                - ovaries
                - corpus luteum
                - placenta
                - foetus
            - functions
                - softens collagen fibres in cervix and ligaments
                - increases uterine blood flow
                - promotes growth of the uterus and breast glandular tissue
                - increases production of insulin-like growth factors
                - enhances myometrial contractility
                - increases myometrial sensitivity to oxytocin
- physiological changes in pregnancy
    - endocrine
        - pituitary hormones
            - FSH/LH fall to extremely low levels due to high levels of oestrogen and progesterone
            - ACTH and melanocyte-stimulating hormone increase
            - prolactin levels increase

- pituitary growth hormone levels fall but overall serum levels increase due to placental production
- oxytocin levels increase to a peak at term
- ADH levels are unchanged

- thyroid and parathyroid gland
  - thyroxine-binding globulin concentrations rise due to increased oestrogen levels
  - T4 and T3 increase over the first half of pregnancy but there is a normal to slightly decreased amount of free hormone due to increased TBG
    - normal ranges are slightly reduced in the second and third trimester
  - TSH production is stimulated after the first trimester
  - foetal need for calcium is high, but maternal serum levels are maintained by increased intestinal absorption
    - there is increased excretion in the urine, with increased risk of renal stones
    - serum vitamin D levels rise
- adrenal gland and pancreas
  - cortisol levels increase, favouring lipogenesis and fat storage
  - insulin response increases, so blood sugar should remain normal or low
  - peripheral insulin resistance increases after early stages of pregnancy
  - the combination of insulin resistance and relatively low glucose promotes the use of fat for energy, preserving glucose and amino acids for the foetus

- cardiovascular system
  - there is peripheral vasodilatation
  - cardiac output increases by 20% by week 8 and then further up to a 40% increase, maximal at week 20-28
    - there is further increased cardiac output in labour and a huge increase immediately after delivery, followed by a return to normal within around an hour
  - there is increased stroke volume and increased heart rate of around 10-20bpm
  - blood pressure is lower than normal in the first two trimesters but returns to normal in the third
  - venous return in the inferior vena cava can be compromised in late pregnancy if the woman lies flat on her back due to pressure from the uterus – reduced cardiac output can compromise foetal blood supply
  - there is increased risk of pulmonary oedema if there is an increase in blood volume or increased pulmonary capillary permeability secondary to pre-eclampsia
  - cardiac examination
    - there may be a bounding or collapsing pulse
    - there may be a third heart sound after mid-pregnancy

        - diastolic murmurs should be considered potentially pathological
        - systolic flow murmurs are common
    - ECG changes considered normal during pregnancy
        - left axis deviation
        - small Q waves and inverted T wave in lead III
        - ST depression and inversion or flattening of the T wave in inferior and lateral leads
        - atrial and ventricular ectopics
- respiratory system
    - tidal volume increases by around 200ml, increasing vital capacity and decreasing residual volume
        - in later pregnancy, splinting of the diaphragm may occur with some decrease in tidal volume
        - respiratory rate does not alter significantly
    - increased oxygen consumption (by around 20%) and increased metabolic rate cause increased oxygen demand
    - state of compensated respiratory alkalosis – arterial pCO_2 drops, arterial pO_2 rises and bicarbonate decreases
        - lower maternal pCO_2 facilitates oxygen/carbon dioxide transfer to and from the foetus
    - many women complain of feeling short of breath, the mechanism is not fully understood
- alimentary system
    - nausea and vomiting are common in early pregnancy
    - appetite is usually increased, sometimes with specific cravings
    - progesterone causes relaxation of the lower oesophageal sphincter and increased reflux, contributed to by pressure from the uterus in later pregnancy
    - gastrointestinal motility is reduced and transit time is longer, allowing adequate nutrient absorption
        - constipation is common
    - the gallbladder may dilate and empty less completely
        - pregnancy also predisposes to precipitation of cholesterol gallstones
    - gums become spongy, friable and prone to bleeding and good dental hygiene is important
- urinary tract changes
    - increased blood volume and cardiac output cause a 50-60% increase in renal blood flow and GFR
    - mild glycosuria and/or proteinuria may occur
    - increased water retention causes reduction of plasma osmolality
    - the smooth muscle of the renal pelvis and ureter become relaxed and dilated, the kidneys increase in length and the ureters become longer and more curved with an increase in residual urine volume
    - bladder smooth muscle relaxes, increasing capacity and risk of UTI
    - the enlarging uterus may put pressure on the ureters
    - 2-10% of women have asymptomatic bacteriuria in pregnancy, and if untreated up to 30% may develop acute pyelonephritis

- haematological changes
    - plasma volume increases by about 50% over the course of the pregnancy, causing dilutional anaemia
    - elevated EPO levels increase the total red cell mass by the end of the second trimester but haemoglobin levels never reach pre-pregnancy levels
    - MCV is usually unaffected
    - there is a modest leucocytosis
    - a normal pregnancy creates a demand for around 1000mg of additional iron (equivalent to 300mg ferrous sulphate/day)
    - levels of some clotting factors increase whilst fibrinolytic activity decreases
        - this protects from haemorrhage at delivery but also makes pregnancy a hypercoagulable state
    - serum albumin decreases
- metabolic changes
    - basal metabolic rate increases slowly over the course of pregnancy, by 15-20%
    - energy requirement does not increase in the first and second trimesters, and increases by around 200kcal/day in the third
    - active energy expenditure tends to fall
    - recommended normal weight gain is11.4 to15.9kg for a woman of normal BMI
        - around 5kg is the foetus, placenta, membranes and amniotic fluid and the rest is maternal stores of fat and protein and increased intra- and extra-vascular volume
- skin
    - hyperpigmentation of the umbilicus, nipples, abdominal midline (linea nigra) and face
    - hyperdynamic circulation and high levels of oestrogen may cause spider naevi and palmar erythema
    - striae gravidarum are common
- musculoskeletal changes
    - increased ligamental laxity caused by increased levels of relaxin contribute to back pain and pubic symphysis dysfunction
    - shift in posture with exaggerated lumbar lordosis leading to the typical gait of late pregnancy

ObP4 genital injury/assault

- external genital trauma
    - blunt scrotal trauma
        - 85% of scrotal injuries are from blunt trauma, usually associated with athletic activity
        - injuries range from bruising of the scrotum through haematocoele to overt testicular rupture
        - testicular torsion and dislocation are rare
        - testicular rupture
            - the tunica albuginea is torn, with evisceration of testicular tubules, mandating urgent surgical repair

                - diagnosis may be clinical by palpation or radiologically by ultrasound
            - guidelines recommend early scrotal exploration in all cases of testicular rupture, torsion and dislocation and with large symptomatic hydrocoeles (due to increased risk of delayed intervention and orchidectomy in those treated conservatively)
    - penile fracture
        - uncommon
        - occurs due to excessive bending of the erect penis, usually during vaginal intercourse
        - a tear of the tunica albuginea of the corpus cavernosum occurs, leading to immediate penile pain and rapid detumescence
        - there is often an audible pop or cracking sound
        - there is swelling and bruising confined to the penile shaft, often described as an aubergine deformity
        - if Buck's fascia is torn, blood may extravasate along facial planes into the scrotum, perineum and occasionally suprapubic areas
        - usually diagnosed on history and clinical examination, with MRI used in equivocal cases
        - management requires prompt surgical repair, usually involving a distal circumferential penile incision with degloving of the penile skin to the location of the injury followed by primary repair of the tunica albuginea defect
- straddle injury in children
    - background
        - most are minor
        - can cause great anxiety due to location and concern for future gynaecological and sexual development
        - unoestrogenised prepubertal female tissues are friable with excellent blood flow and lack distensibility so even minor trauma can cause injury and bleeding which may appear extensive
    - history
        - mechanism of injury
        - timing and setting of injury
        - first aid provided
        - ability to pass urine and faeces
        - other injuries
        - witnesses
        - any inconsistencies or suspicion of non-accidental injury
    - examination
        - general examination
        - genital examination should be performed only once (ideally with senior clinician)
            - if suspicious for non-accidental injury the forensic medical service should be involved
            - examination is usually in the supine frog leg position
            - explanation and gentle handling is important and consideration should be given to analgesia +/- procedural sedation

                - a detailed description of the injury should be documented using clock face notation
            - features to consider on examination
                - can the posterior or upper limit of the wound be seen? – if not, examination under anaesthesia should be considered
                - is there an expanding haematoma?
                - is there any anal or rectal involvement?
        - management
            - general principles
                - forensic involvement if suspicious for non-accidental injury
                - compression of bleeding with a clean dressing pad
                - if there is significant vaginal bleeding in older adolescents, the vagina can be packed with a tampon or gauze
                - ice packs to reduce bleeding and swelling (avoiding directly over the clitoris)
                - irrigation with warm water
                - review of tetanus status
            - minor injury (bleeding is minor or has stopped and the child can void spontaneously)
                - salt water baths for comfort
                - topical anaesthetic cream or barrier cream to reduce local pain on micturition
                - reduction in strenuous activity for 24 hours to prevent re-injury
                - simple analgesia
                - follow up with GP
            - non-minor (ongoing bleeding, laceration borders not visualised. labia minora tear, unable to void, clinician concern)
                - management as above
                - consider urethral catheter if unable to void
                - follow up with local paediatric team or gynaecology/surgical team as indicated
- sexual assault (RCEM guidance)
    - where possible and practical, victims of sexual assault and rape should be assessed in a Sexual Assault Referral Centre (SARC)
        - the SARC is a one stop location where victims can receive a forensic examination, emergency contraception and post-exposure prophylaxis, psychological counselling, legal advice and support
        - victims can choose to be dealt with anonymously if they do not want police involvement
    - any forensic examination should only be performed by a clinician with suitable specialist training in an appropriate environment
        - pelvic examination should not be performed in the ED unless there is significant bleeding
        - the forensic medical examiner should be able to attend the hospital if the victim requires further hospital management
        - ED staff should wear gloves and take care not to cut through blood stains/knife holes etc.

- personal identifiable information about sexual assaults and rapes should not normally be shared without consent, except in exceptional circumstances
- emergency contraception should be available to victims attending the emergency department
- there is no requirement for emergency physicians to take pre-transfusion blood samples for the police
    - if required, it should be taken by an appropriately trained person, usually an FME, to ensure chain of evidence
- post-exposure prophylaxis for sexually transmitted infections should be available to victims attending the emergency department
    - the donor is rarely available or willing to provide samples
    - baseline blood tests should be taken for Hep B, Hep C and HIV in the ED
    - if the perpetrator is not known to be Hep B negative, accelerated vaccination should be considered, with Hep B immunoglobulin in high risk situations
    - PEPSE for HIV should be considered when:
        - the assailant is known to be HIV positive
        - the assailant had risk factors
        - anal rape
        - multiple assailants
        - bleeding
            - PEPSE may be effective if administered within 72 hours but is more effective when administered earlier
            - EDs should have access to PEPSE, and patients should be followed up by the appropriate service
    - transmission of bacterial infections is more likely when there are:
        - multiple assailants
        - biting
        - defloration
        - wounds
        - anal intercourse
        - where the assailant is a man who has sex with men or injects illegal drugs
            - prophylactic antibiotics should be offered (usually cefixime 400mg, azithromycin 1g and metronidazole 2g as a single oral stat dose)
- the role of the emergency physician:
    - be sensitive and sympathetic, and aware that absence of injuries does not exclude sexual assault
    - take a careful history of the assault including time and location, characteristics of the assailant(s), physical violence, sexual acts (vaginal, oral, anal; penile or digital penetration)
    - particular effort should be made to take accurate and contemporaneous notes
    - any forensic examination should only be undertaken by a physician with specialist training, ideally in a forensically secure environment

        - the medical examination should ideally be performed by the most senior or appropriately trained doctor in the ED; patients should be offered a choice of gender of doctor where possible
        - examine everywhere including inside the mouth looking for bruising, abrasions and lacerations and any patterns such as fingertip marks; look for and treat any bite marks
        - document examination findings clearly as you may be asked to produce a statement or go to court
        - consider the possibility of pregnancy; emergency contraception should be offered
        - consider the possibility of exposure to sexually transmitted infections
        - victims may need further referral to Social Services, Victim Support, a Community Safety unit or other organisations which can offer support
        - any concerns about child welfare or vulnerable adults should lead to the activation of local safeguarding procedures
    - information sharing with other agencies
        - it should be assumed that clinical information is confidential
        - patients should be offered police involvement, but a decision not to should be respected
        - in exceptional circumstances, information can be shared with the police and social services, e.g.:
            - where the victim is a child – local safeguarding procedures should be triggered
            - where there are concerns about the children of the victim
            - where the victim lacks capacity and is unlikely to regain capacity
            - where guns or knives have been used by the perpetrator
        - a decision to share should be taken by the consultant and ideally discussed with another consultant; clear documentation of the decision to share should be documented and explained to the patient where possible

ObP5 vaginal discharge

- background
    - common presenting complaint
    - normal physiological discharge is a white or clear, non-offensive discharge that varies with the menstrual cycle
    - the most common cause of pathological vaginal discharge in women of childbearing age is bacterial vaginosis
    - vulvovaginal candidiasis affects around 75% of women at some point during their reproductive life
- causes
    - non-infective
        - physiological
            - newborn infants may have a small amount of vaginal discharge, sometimes mixed in with a little blood, due to high levels of circulating maternal oestrogen
                - should disappear by 2 weeks of age

- during the reproductive years, fluctuating levels of oestrogen and progesterone throughout the menstrual cycle affect the quality and quantity of cervical mucus which is perceived as a change in vaginal discharge
- at menopause the normal amount of vaginal discharge decreases as oestrogen levels fall

- cervical polyps and ectopy
  - tend to be asymptomatic, may have increased discharge and intermenstrual bleeding
  - diagnosed on speculum examination
  - polyps can be removed and sent for histology
- foreign bodies – e.g. retained tampon
  - foul-smelling serosanguinous discharge
  - diagnosis confirmed on examination
  - most can be manually removed, some may require lavage (+/- sedation in children)
- vulval dermatitis
  - apparent on examination, requires biopsy to confirm
- erosive lichen planus
  - apparent on examination, requires biopsy to confirm
- genital tract malignancy – e.g. cancer of the cervix, uterus or ovary
  - presentation varies
  - persistent vaginal discharge not responding to conventional discharge may be the only clue
  - diagnosis is made on examination and biopsy
- fistulae
  - history of trauma or surgery is suggestive
  - there may be foul or feculent discharge in association with recurrent UTIs
- allergic reactions
  - use of irritant chemicals in douching, contact with latex and semen

- non-sexually-transmitted infection
  - bacterial vaginosis
    - most commonly seen in sexually active women
    - causes thin, profuse, fishy-smelling discharge without itch or soreness
  - candida infections
    - thick, curd-like, white, non-offensive discharge associated with vulval itch and soreness; may cause mild dyspareunia and dysuria
- sexually-transmitted infection
  - Chlamydia trachomatis
    - may cause copious purulent vaginal discharge but is asymptomatic in 80%
  - Neisseria gonorrhoeae
    - may have a purulent vaginal discharge; asymptomatic in up to 50%
  - Trichomonas vaginalis

        - may cause an offensive yellow discharge, often profuse and frothy, associated with vulval itch and soreness, dysuria, abdominal pain and superficial dyspareunia
- assessment
    - full clinical and sexual history
    - features of discharge:
        - odour
        - onset
        - duration
        - colour
        - consistency
        - associated symptoms
            - itch
            - superficial dyspareunia or dysuria
            - abdominal pain
            - deep dyspareunia
            - abnormal bleeding
            - pyrexia
    - assessment of risk of STI
    - current contraceptive use and concurrent medications, previous treatments
    - medical conditions
    - symptoms suggesting that discharge is abnormal include:
        - a discharge that is heavier than usual
        - a discharge that is thicker than usual
        - pus-like discharge
        - white and clumpy discharge
        - greyish, greenish, yellowish or blood-tinged discharge
        - foul smelling (fishy or rotting meat) discharge
        - discharge accompanied by bloodiness, itching, burning, rash or soreness
- investigations
    - may not be required if diagnosis clear and patient low risk for STIs
    - STI testing
    - vaginal pH testing
- management
    - education and counselling as required
    - metronidazole and clindamycin for BV
    - vaginal or oral azole antifungals for candidiasis
- complications
    - untreated, some vaginal infections can spread to the upper reproductive tract and cause more serious illness, and infertility in the long term
    - retained foreign body may lead to toxic shock syndrome
    - cervical polyps may cause infertility if very large
        - they may rarely undergo malignant change
- prognosis
    - BV has a 70-80% cure rate after one course of treatment, but often recurs
    - candidiasis has a cure rate of 80-95%
    - trichomoniasis has an approximate 90% cure rate

- prevention
    - basic personal hygiene
    - avoidance of douches and perfumed chemical agents
    - avoidance of tight synthetic clothing
    - emollients can be used as a soap substitute
    - treatment of sexual partners as appropriate

ObP6 foreign bodies

- vaginal foreign bodies
    - background
        - present in females of all ages
        - in paediatrics, the most common objects are toys, tissue paper and household objects
        - in adult women, they include tampons, condoms, menstrual cups and items used for sexual gratification
        - elderly patients are at increased risk for retained medical devices such as pessaries
    - presentation
        - may self-report
        - may present with a variety of symptoms such as:
            - pelvic pain
            - vaginal discharge
            - vaginal bleeding
        - history should include:
            - timing
            - suspected object
            - symptoms of the abdomen, pelvis and genitalia
        - sexual abuse should be considered, particularly in the paediatric population
    - examination
        - a chaperone must be present, and if possible the patient should be offered a female chaperone
        - includes a genital examination
            - may include speculum examination
        - imaging is an option if the diagnosis is unclear or complications are suspected
            - e.g. x-ray, CT, MRI, US
                - transabdominal US is preferred in paediatric patients due to low radiation and invasiveness
        - paediatric patients and patients with behavioural disturbance may require examination under sedation once appropriate consent has been obtained
        - pelvic examination should not be done if there is suspicion of assault
    - management
        - many foreign bodies can be removed with forceps under direct visualisation during the speculum examination
            - a non sterile. lubricated speculum should be used with a reliable light source
            - culture swabs should be available if discharge is reported or seen

        - surgical intervention may be required in certain cases such as:
            - foreign bodies in place for an extended period
            - size, position, location
        - general anaesthesia can allow for full muscle relaxation
    - complications
        - infection
        - compromised blood flow due to compression on surrounding tissues
            - can lead to perforation and fistulae
        - ulceration
            - particularly if caustic objects involved, such as batteries
        - toxic shock syndrome
            - usually related to super absorbent tampons but may be associated with other sources such as menstrual cups
            - patients present with fever, rash, desquamation and sepsis
        - migration of sharp foreign bodies such as needles
- urethral/penile foreign body
    - rare
    - radiological evaluation is required to determine size, location and number of foreign bodies
    - endoscopic retrieval is usually successful and antibiotic coverage is necessary
    - psychiatric evaluation is recommended
    - can lead to urethral stricture and follow up is recommended

ObP7 patient in labour

- normal labour
    - first stage
        - begins with regular contractions (when the foetal presenting part has descended into the true pelvis)
        - the first stage ends when the cervix is fully dilated (10cm)
        - can be divided into:
            - latent or quiet phase
                - contractions are not particularly painful and at 5-10 minute intervals
                - contractions become stronger with shorter intervals, although the cervix is still dilating relatively slowly, with membranes possibly breaking later in this phase
            - active phase
                - starts with the cervix 3-4cm dilated and is associated with more rapid dilatation (normally 0.5-1cm/hr)
                - once the cervix is dilated to 9cm, towards the end of the active phase, contractions may be more painful and the woman may want to push
                - pushing is undesirable at this stage; there is the need to establish by vaginal examination whether the cervix is fully dilated
                - during this time the foetal head descends into the maternal pelvis and the foetal neck flexes

        - the length of established first stage of labour varied between women, but lasts on average 8 hours (unlikely to be longer than 18 hours); second and subsequent labours last on average 5 hours
        - if the first stage does not appear to be progressing, the cause needs to be determined
        - management
            - reassure and advise the woman on how her labour is progressing
            - pulse should be measured hourly, and temperature and blood pressure 4-hourly
            - monitor frequency of contractions half hourly
            - foetal heart rate should be auscultated for at least 1 minute following a contraction – this should be carried out every 15 minutes, with maternal pulse palpated to differentiate
            - foetal heart rate should be 100-160bpm
            - a vaginal examination to assess cervical dilatation and foetal head descent should be offered every four hours and when the woman appears to be in established labour
            - discuss the patient's need and plan for pain relief
            - assess the position of the foetal head with regards to the maternal pelvis
            - a partogram (pictorial record of labour) should be used once labour is established
                - the WHO recommends a four hour action line, meaning that if labour does not progress as anticipated, action will be taken (e.g. amniotomy or augmentation)
    - second stage
        - starts when the cervix is fully dilated and ends with the birth of the baby
        - contractions are stronger, occur at 2-5 minute intervals and last 60-90 seconds
        - the foetal head descends deeply into the pelvis and rotates anteriorly so that the back of the foetal head is behind the mother's symphysis pubis
        - the second stage is said to be active once the baby is visible and the woman usually also wants to assist what have become expulsive contractions by pushing
        - the foetal head becomes more visible with each contraction until a large part of the head can be seen
        - the head is now born with first the forehead, then the nose, mouth and chin
        - the head rotates to allow the shoulders to be born next, followed by the trunk and legs
        - after this, the baby should start to breathe and cry loudly
        - management
            - check level of pain and supplement relief if required
            - ensure a midwife/doctor is present at all times to encourage pushing during contractions and relaxing in between
            - monitor contractions and foetal heart rate (every five minutes, should be 100-160bpm)

- if this stage is >2 hours for a nulliparous woman or >1 hour for a multiparous woman, instrumental delivery should be considered
- there is debate about the optimal method to use during the second stage to reduce perineal trauma:
    - hands on, where pressure is placed on the baby's head and the perineum supported; application of a warm compress appears to reduce the severity of perineal trauma
    - hands poised, where these manoeuvres are not carried out; may reduce episiotomy rates but insufficient evidence
- position during the second stage
    - no good evidence exists to suggest optimal position for labour, so women should be encouraged to adopt the position they find most comfortable

- third stage
    - starts with the birth of the baby and ends with the delivery of the placenta and membranes
        - separation of the placenta occurs immediately after birth due to forceful uterine contractions along with retraction of the uterus, thus greatly reducing the size of the placental bed
        - it normally takes up to 5 minutes, but can take longer
        - haemorrhaging is prevented by the contraction of uterine muscle fibres closing off the blood vessels that were supplying the placenta
        - without active management, after 10-20 minutes, separation is shown by a gush of blood, prominence of the fundus in the abdomen and apparent lengthening of the umbilical cord
    - management
        - expectant (traditional or physiological)
            - once the placenta lies in the vagina, the uterus is 'rubbed up' to produce a contraction and the uterus is pushed towards the vagina to help with expulsion of the placenta and membranes
            - these are held and twisted whilst pulling constantly so that membranes are kept intact
            - the cord is not clamped until pulsation has stopped and no uterotonic drugs are used
            - should last <60 minutes
        - active
            - IM synthetic oxytocin is given with the delivery of the anterior shoulder or as soon as the baby is born
            - the umbilical cord is clamped between 1-5 minutes after the birth and cut soon after delivery
            - after the cord has been cut and once there are signs of separation of the placenta, controlled traction on the umbilical cord, with simultaneous suprapubic pressure by the other hand to prevent uterine inversion, will facilitate expulsion of the placenta and membranes

        - in a small proportion of cases, the placenta is not removed – repeat the attempt after 10 minutes then remove manually
        - in all cases, the placenta and membranes are examined for completeness and any retained material removed under anaesthetic
        - should last <30 minutes
    - active management has been shown to be superior with respect to blood loss, blood transfusion, postpartum haemorrhage and other serious complications of the third stage
        - if ergometrine is used, there is an increased incidence of side effects such as nausea, vomiting and hypertension – 10IU of IM oxytocin are the preferred uterotonic
        - active management should be offered routinely for vaginal deliveries in a hospital setting, however a woman who is at low risk of postpartum haemorrhage and who requests physiological management should have her request respected
  - there is good evidence that women who receive continuous one-to-one support throughout their labour have better outcomes in terms of reduced analgesia requirement, decreased frequency of operative delivery and improved satisfaction
    - particularly when the supporter or doula was not a member of hospital staff, gave support from early in labour and where epidural analgesia was not routinely available
- Emergency Department management of labour in concealed pregnancy
  - check the diagnosis
    - obesity can obscure the 'baby bump', an anterior placenta can mean that not many foetal movements are felt, and denial can be strong
    - ultrasound can be helpful unless the head is already in the pelvis
    - look for an emerging head
  - get help
    - midwife, obstetrician, neonatologist
    - in a concealed pregnancy you do not know the gestation or position of the baby and there has not been any screening for congenital abnormality
  - prepare
    - split the team into two
    - if the baby is preterm, it is important that the team knows how to deal with this
    - get the resuscitaire and turn on the heater, attach oxygen and prepare equipment
    - prepare towels and a cord clamp
    - incontinence pads help to estimate blood loss
    - maternity pad or sanitary towel to catch and wipe away stool
  - reassurance
    - the mother is dealing with a lot
    - allocate a nurse to support her
    - checking obs repeatedly is not required
    - a cannula is probably not required
  - analgesia

        - provide Entonox
        - take it away during pushing, then provide it between pushes
    - delivery
        - once you see the head, encourage pushing until the head is out
        - support the head as it turns to face the side
        - guide the head downwards on the next contraction to allow delivery of the anterior shoulder, then upwards to allow delivery of the posterior shoulder – all should be very gentle, just taking the weight and supporting
        - baby should deliver quickly after this
    - assess baby
        - wipe away any vernix from the face, wrap it in a towel and give a quick rub
        - transfer with cord attached to mum's abdomen and chest for skin to skin time
        - if they do not pink up and cry immediately, wipe away any vernix and continue to stimulate by rubbing with a towel
        - if baby remains blue, floppy and unresponsive, clamp and cut the cord and take the baby to the resuscitaire
            - put two clamps in the middle of the cord and cut between them – allows enough length for insertion of umbilical lines later if needed
    - deliver the placenta
        - can usually be left for the obstetrics team to deliver later
        - if not possible, give 500mcg/1ml IM Syntometrine as soon as possible after labour
        - during the first strong contraction, you may notice a small rush of blood and lengthening of the cord as the placenta separates from the uterus
            - a little controlled traction on the cord will deliver the placenta
            - do not pull too hard, as this can cause uterine inversion
- active management of labour
    - initially developed to reduce prolonged labours and try to keep them under 12 hours, with minimal operative delay rates
    - originally designed for primiparous women with singleton pregnancies at term, in spontaneous labour
    - core principles:
        - early diagnosis following strict criteria, by a senior midwife
        - vaginal examination hourly for three hours, then every two hours at least, allowing rate of progression to be plotted on a partogram
        - amniotomy one hour after admission
        - augmentation with Syntocinon if not dilating at 1cm/hour
        - women not in labour should be sent home
        - personal, psychological support for the woman
        - liberal use of epidural anaesthesia
        - regular rounds by the obstetrician
        - early cord clamping and controlled cord traction with uterotonic
        - antenatal education classes
        - regular audit of labour ward processes and outcomes
- cord prolapse
    - three types
        - overt cord prolapse

                - if the presenting part does not fit the pelvis snugly after membrane rupture, the cord can slip past and present at the cervix or descend into the vagina
                - it is an acute obstetric emergency, as prolapse exposes the cord to intermittent compression compromising the foetal circulation
                - depending on duration and degree of compression, foetal hypoxia, brain damage and even death can occur
                - exposure of the umbilical cord to air causes irritation and cooling, resulting in vasospasm of the cord vessels
            - occult cord prolapse
                - where the cord lies alongside the presenting part
            - cord presentation
                - the cord can be felt to prolapse below the presenting part with or without membrane rupture
                - the cord may slip to one side of the head and disappear as the membranes rupture
                - when the cord can be felt to prolapse below the presenting part before membranes have ruptured, this is referred to as funic presentation
    - risk factors
        - prematurity
        - foetal congenital abnormality
        - second twin
        - multiparity
        - low birth weight
        - breech
        - oblique, transverse and unstable lie
        - cephalopelvic disproportion
        - pelvic tumours
        - low-lying placenta
        - polyhydramnios
        - macrosomia
        - high foetal station
        - long umbilical cord
        - obstetric interventions including:
            - amniotomy with high presenting part
            - vaginal manipulation of the foetus with ruptured membranes
            - insertion of intrauterine pressure catheter
            - attempted external cephalic or internal podalic version
    - presentation
        - may have no outward signs and only be detected by foetal monitoring – foetal decelerations during contractions which return to normal after the contraction, bradycardia with prolonged and complete compression
    - emergency management
        - urgent transfer to consultant-led obstetric unit
        - advise knee-chest position (patient facing the bed, chest level to bed, knees tucked under chest, pelvis and buttocks elevated)

        - elevate the presenting part (two fingers or hand pushing upwards against the presenting part or, once the presenting part is above the pelvic brim, using continuous suprapubic pressure in an upwards direction
            - can also be achieved by filling the urinary bladder
        - manual replacement of the prolapsed cord above the presenting part is not currently recommended
            - handling of the cord outside the vagina induces vasospasm
        - the patient requires emergency c-section as soon as possible
        - if available, terbutaline 0.25mg SC can be given to reduce contractions when there are persistent foetal heart rate abnormalities despite attempts to prevent cord compression manually, and there may be delays in achieving delivery
        - vaginal delivery should only be continued if delivery is imminent, the cervix is fully dilated and there are no contra-indications
            - episiotomy/vacuum extraction or forceps can expedite delivery
        - transfer should be in the left lateral position with a pillow under the hip

ObC1 ante-partum haemorrhage

- definition:
    - bleeding after 24 weeks gestation
    - 2.5% of pregnancies
- causes:
    - placental abruption
    - placenta praevia
    - uterine rupture
    - bleeding from lower genital tract
        - cervicitis, cervical cancer or polyp, vaginal cancer
    - bleeding that may be confused with vaginal bleeding
        - haemorrhoids, inflammatory bowel disease, UTI
- placental abruption:
    - premature separation of part of the placenta from the uterus
    - outcome depends on degree of separation and amount of blood loss
    - risk factors:
        - pre-eclampsia
        - previous placental abruption
        - trauma
        - smoking
        - multi-parity
    - clinical features:
        - vaginal bleeding
            - amount depends on location and degree of separation
            - can be concealed haemorrhage
        - abdominal pain and tenderness
        - premature labour
        - haemodynamic compromise
            - may be out of proportion to degree of visible blood loss
        - DIC or absent foetal heart sounds in large bleeds

- placenta praevia
    - placenta inserted wholly or partially in the lower segment of the uterus
    - if it lies over the cervical os it is a major praevia, if not it is minor
    - risk factors include:
        - maternal age >35
        - high parity
        - previous placenta praevia
        - twin pregnancy
        - uterine abnormalities
            - e.g. previous c-section, fibroids
    - clinical features:
        - bright red, painless vaginal bleeding in third trimester
        - labour (in 15%)
- vasa praevia
    - rare condition where foetal blood vessels grow within the membranes and over the internal os
    - haemorrhage may occur when the membranes rupture, resulting in foetal exanguination
- investigations:
    - FBC, clotting, fibrinogen (risk of DIC)
    - cross match 6 units
    - Rhesus status (may need anti-D)
    - Kleihauer test (to determine degree of foetal-maternal haemorrhage)
- management:
    - manage in resus
    - call obstetrician immediately
    - two large bore cannulae and fluid resuscitation
    - blood transfusion as required
    - anti-D immunoglobulin if Rhesus-negative

ObC2 bleeding in early pregnancy

- causes of bleeding in pregnancy
    - 1st trimester
        - spontaneous miscarriage
        - ectopic
        - trophoblastic disease
    - 2nd trimester
        - spontaneous miscarriage
        - trophoblastic disease
        - placental abruption
        - placenta praevia
    - 3rd trimester
        - placental abruption
        - placenta praevia
        - vasa praevia
        - 'show' of pregnancy
- spontaneous miscarriage

- loss of pregnancy before 24 weeks gestation
- occurs in around 1 in 5 pregnancies
- most present in the first 12 weeks with vaginal bleeding and/or abdominal pain
- terms:
    - threatened miscarriage
        - vaginal bleeding through a closed cervical os
        - 50% proceed to miscarriage
    - inevitable miscarriage
        - vaginal bleeding through an open os or passage of some products of conception
    - incomplete miscarriage
        - vaginal bleeding but not all products of conception have passed
    - missed miscarriage
        - failure of the pregnancy is 'silent' and often found on ultrasound
    - miscarriage with infection
        - occurs when retained products become infected
    - complete miscarriage
        - once all products of conception have passed
- clinical features of spontaneous miscarriage:
    - vaginal bleeding
        - from light spotting to heavy bleeding with clots
    - abdominal pain
        - usually lower abdomen, crampy
        - unilateral pain or shoulder tip pain (diaphragmatic irritation) suggest ectopic
    - cervical shock
        - products of conception become stuck in the cervical os causing profound vagal stimulation (hypotension and bradycardia)
        - severe abdominal pain
    - vaginal examination
        - may reveal an open os indicating an inevitable miscarriage
        - cervical or adnexal tenderness indicates possible ectopic or infection
- investigations:
    - urinary pregnancy test
        - may remain positive for several days after foetal death
    - urine dipstick
        - to check for infection which may account for lower abdominal pain
    - serum β-hCG
        - can be helpful for serial monitoring, levels would fall on serial testing in a miscarriage
    - ultrasound
        - earliest a gestational sac can be seen via transabdominal scan is 7 weeks
        - earliest via transvaginal scan is 5-6 weeks
    - Rhesus status check
    - FBC< clotting, cross-match if shocked
- management:

        - fluid resuscitation as required
        - analgesia
        - removal of products of conception from os if in cervical shock
        - ergometrine (500 microgram IM) if severe bleeding
        - urgent referral to gynaecology if haemodynamically compromised or ectopic suspected
        - referral to Early Pregnancy Unit as outpatient if stable
- gestational trophoblastic disease
    - fertilised ovum forms abnormal trophoblastic tissue but no foetus
    - spectrum from benign (hydatidiform mole) to malignant (choriocarcinoma)
    - excessive production of β-hCG from abnormal placental tissue, may cause exaggerated symptoms of early pregnancy
    - 50% have vaginal bleeds at 12-16 weeks which may be heavy and include passage of molar tissue which resembles frog spawn
    - ultrasound shows 'snowstorm' and no foetus
    - serum β-hCG is grossly elevated
    - ED management involves IV fluids, analgesia and referral to gynaecology for evacuation of the uterus

ObC3 exposure to infections during pregnancy e.g. chickenpox

- parvovirus B19
    - background
        - pregnant women who develop erythema infectiosum have a 30% chance of passing it on to their unborn baby and a 5-10% chance of foetal loss
            - risk is heightened in the first trimester
        - maternal symptoms are usually short lived
        - foetal complication include:
            - hydrops fetalis
            - hepatitis
            - severe anaemia
            - inflammation of the heart
            - cardiac failure
        - intrauterine transfusion improves outcome
    - investigation and management
        - all pregnant women with a non-vesicular rash, or contact with someone with a non-vesicular rash, should be investigated for parvovirus and rubella, irrespective of past history, previous serology or gestation
            - contact is defined as being in the same room for >15 minutes or face to face contact
            - main risk of infection for parvovirus is from household contacts or prolonged occupational contact
        - IgM result confirms or excludes infection in the four weeks prior to the sample
            - parvovirus cannot be excluded if investigation starts >4 weeks after onset of rash
        - testing should be for parvovirus B19 and rubella IgM and IgG as soon as possible after contact with or symptoms of a rash illness

                - positive IgG with negative IgM confirms immunity and the patient can be reassured
                - if IgM is detected but IgG is not, a further sample should be taken immediately; if still positive for IgM, confirmation by an alternative assay is required
                - if neither IgG nor IgM is detected, a further sample should be tested one month later
                    - if both are negative, the woman can be reassured but she remains susceptible
            - confirmed parvovirus infection
                - the patient should be referred to a foetal medicine unit capable of foetal blood sampling and transfusion
                - an earlier blood sample should be tested if possible
                - another serum sample should be taken 7-10 days later to confirm the finding
                - serial ultrasound and Doppler assessment of the foetus are usually undertaken
                - early delivery may be necessary if the foetus is near full term
- rubella
    - background
        - viral infection causing a pink rash with swelling of the lymph glands behind the ears and at the back of the head
        - mild constitutional symptoms and occasionally joint pain in adults
    - consequences
        - if contracted within the first 11 weeks of pregnancy there is a 90% chance of the foetus being infected, falling to 20% at week 11-16 and further after that
        - foetal defects include:
            - general learning disability
            - cataracts
            - deafness
            - heart defects
            - intrauterine growth restriction
            - inflammation of the brain, liver, lungs and bone marrow
    - management
        - testing for rubella and parvovirus B19 with any rubella-like rash (even if reported immune) – serum for IgG and IgM with repeat if equivocal or advised by lab
        - evidence of infection should be discussed with the patient with a view to considering termination if indicated and appropriate, in consultation with a foetal medicine specialist
        - rubella is a notifiable disease
- chickenpox (varicella zoster)
    - background
        - characterised by fever, malaise and a pruritic rash that develops into crops of maculopapules, which become vesicular and crust over before healing
        - incubation is 7-21 days and the disease is infectious 48 hours before the rash appears until all vesicles crust over (usually 5-6 days)

        - contact with chickenpox in pregnancy is common, but more than 90% of the population are seropositive for IgG antibody, so primary infection is uncommon (estimated 3 in 1000 pregnancies)
    - consequences
        - greater morbidity in adults – pneumonia (10% of pregnant women), hepatitis and encephalitis
        - may cause foetal varicella syndrome
            - before 20 weeks of gestation
                - skin scarring in a dermatomal distribution
                - microphthalmia, chorioretinitis, cataracts
                - hypoplasia of the limbs
                - neurological abnormalities
                    - e.g. microcephaly, cortical atrophy, intellectual disability, dysfunction of bowel and bladder sphincters
            - 20-36 weeks of gestation
                - does not appear to be associated with adverse effects
                - may present as shingles in the first few years of infant life
            - after 36 weeks
                - up to 50% of babies are infected and around 23% develop clinical varicella despite maternal antibodies
                - most severe chickenpox occurs if the infant is born within 7 days of onset of the mother's rash
    - management
        - of exposure
            - establish whether the mother is immune
                - immunity can be assumed if there is a history of chickenpox or shingles
            - if there is any doubt, urgent antibody (IgG) levels
            - varicella zoster immunoglobulin (VZIG) may be indicated
        - of chickenpox in pregnancy
            - specialist obstetric advice about need for counselling on risk of foetal complications, antiviral treatment and follow-up
            - VZIG is not helpful in established infection
            - close monitoring for complications
        - admission if:
            - respiratory symptoms
            - neurological symptoms other than headache
            - haemorrhagic rash or bleeding
            - severe disease (e.g. dense rash)
            - significant immunosuppression, including systemic corticosteroids in the previous 3 months
- cytomegalovirus
    - background
        - common viral infection, usually mild or asymptomatic in healthy people
        - may cause a febrile illness or have complications
        - most common congenitally acquired infection

        - can be transmitted via breastfeeding, close contact, sexual activity, blood transfusion and organ transplantation
        - risk of transmission to foetus is around 32% during a primary infection and 1.4% in reactivation or reinfection
    - consequences
        - diagnosis can be made antenatally with amniocentesis at least 7 weeks after infection and after 21 weeks of gestation
            - if infection is confirmed, US is performed regularly for monitoring
        - the effect on the foetus is more severe from a primary infection
        - effects on the foetus or neonate (which may not become apparent until later) include:
            - intrauterine growth restriction and low birth weight
            - hepatosplenomegaly
            - jaundice
            - thrombocytopaenia
            - anaemia
            - hydrops and ascites
            - petechiae and/or purpura
            - microcephaly
            - intracranial calcifications
            - choroidoretinitis
            - deafness
            - speech defects
            - general learning disability, which may appear later
            - psychomotor delay
            - visual impairment
    - management
        - there is no effective therapy
        - vaccination is under development
        - IgG antibody testing can confirm primary infection
        - prevention is the most important strategy, including:
            - thorough handwashing after nappy changing, feeding a child or wiping their face, or handling children's toys
            - not sharing food or utensils with a young child
            - avoid kissing a child on or near the mouth
- measles
    - background
        - rare in the UK, with rising incidence due to vaccine hesitancy
        - clinical features include:
            - disseminated maculopapular rash
            - coryza
            - conjunctivitis
        - complications include:
            - pneumonia
            - otitis media
            - encephalitis
    - consequences

        - uncommon in pregnancy but may be associated with:
            - increased risk of maternal morbidity
            - intrauterine death or premature labour
            - rarely, neonatal subacute sclerosing panencephalitis
    - management
        - testing of IgG if exposure is suspected and there is no history of vaccination or immunity
        - if not detected, human normal immunoglobulin (HNIG) can be given; may not prevent measles but may attenuate the illness
- toxoplasmosis
    - background
        - infection by the parasite Toxoplasma gondii, with the cat as the definitive host
        - primary infection is often subclinical; may have a nonspecific illness – fatigue, headaches, muscle pains, low-grade fever, isolated lymphadenopathy
        - can be confirmed through serological testing, and presence of intrauterine spread by amniocentesis
    - consequences
        - greatest risk of transmission is in the third trimester, but consequences are more severe in the first trimester
        - systemic disease of the neonate
            - rash
            - jaundice
            - thrombocytopenic purpura
            - hepatosplenomegaly
            - pneumonia
            - progressive uveitis
        - neurological disease
            - hydrocephalus – due to stenosis of the duct of Sylvius, requiring shunt
            - microcephaly
            - microphthalmia
            - retinochoroiditis
            - cerebral calcification
        - mild disease
            - a small area of retinochoroiditis or slight cerebral calcification without signs of brain damage
        - subclinical
        - relapsing
            - retinochoroiditis as flare-ups can occur at any age, mostly in a previously intact retina
    - management
        - by a specialist
        - includes spiramycin throughout pregnancy for foetal prophylaxis and regular US

        - where infection is confirmed, pyrimethamine, sulfadiazine and folinic acid are used
- pelvic inflammatory disease
    - background
        - commonly STIs
        - PID presents with lower abdominal pain and tenderness
            - may also present with dyspareunia, abnormal vaginal bleeding and/or discharge
            - many cases are asymptomatic
    - consequences
        - PID is associated with an increase in pre-term delivery and maternal or foetal morbidity
        - ophthalmia neonatorum, which is potentially sight-threatening, can be transmitted
    - management
        - admission for IV antibiotics
            - none of the evidence-based regimens are proven to be safe in pregnancy, but benefit may outweigh risk
            - cephalosporins are likely to be an appropriate choice
            - doxycycline is contraindicated after the fifteenth week due to tooth and bone discolouration and inhibition of bone growth
            - erythromycin and metronidazole are probably safe
- genital herpes simplex
    - background
        - produces patches of small, fluid-filled vesicles that burst to form shallow, painful ulcers
        - initial infection is followed by recurrences, which are self-limiting
        - all cases in pregnancy should be referred to a genitourinary specialist advice and PCR testing
    - consequences
        - HSV in pregnancy may cause infection in the infant, either:
            - congenital herpes due to transfer of infection in utero or
            - neonatal herpes infection which occurs as a result of infection at the time of birth
        - congenital herpes is extremely rare; may affect the skin, eyes and CNS, and there may be foetal growth restriction or foetal death
        - neonatal herpes is very rare but serious with high morbidity and mortality
            - risk of adverse consequences is significantly higher in primary infection
            - greatest risk of transmission is when a new infection is contracted in the third trimester, particularly within six weeks of delivery
            - neonatal infection contracted in labour or postnatally may cause eye, skin or mouth lesions, HSV encephalitis or disseminated HSV with multisystem disease
            - with antiviral treatment, mortality from disseminated disease is 30% and from encephalitis alone it is 6%
    - management

        - depends on whether infection is primary or a recurrence and on gestation
        - oral or IV acyclovir should be considered in primary infection in the first or second trimester
        - daily suppression with oral acyclovir may be used from 36 weeks to reduce the chances of lesions being present at delivery and the need for c-section
        - treatment should be continued until delivery if infection is contracted in the third trimester
            - c-section should be recommended unless antibody tests subsequently confirm a recurrent rather than primary infection
            - in recurrent infection, risk of neonatal herpes is low even if lesions are present at time of delivery
- HIV
    - background
        - transmission to the baby is a significant problem, especially in low income countries
        - all pregnant women are screened for HIV
    - consequences
        - vertical transmission rates have fallen, and mortality rates have declined with treatment; however HIV has serious lifelong consequences
    - management
        - multidisciplinary management involving an HIV physician, an obstetrician, a paediatrician and a specialist midwife
        - transmission can be reduced by:
            - maternal diagnosis and treatment with antiretrovirals
            - appropriate management of delivery – women with low viral load may be safe to deliver vaginally
            - avoidance of breastfeeding
            - neonatal treatment with antiretroviral therapy from birth, followed by a series of HIV tests
- urinary tract infections
    - background
        - asymptomatic bacteriuria is very common in pregnant women because of the altered dynamics of the urinary tract
        - if untreated, it frequently progresses to UTI
    - consequences
        - UTI increases the risk of pyelonephritis and may increase the incidence of premature labour and low birth weight
    - management
        - urine should be screened by sending urine for culture at the first antenatal visit, with a second sample being sent if bacteria found
        - asymptomatic and symptomatic UTI should be treated with antibiotics and urine sent for culture at every antenatal visit until delivery
        - antibiotic should be as per local guidelines – usually amoxicillin, nitrofurantoin, trimethoprim or cefalexin for seven days followed by testing for resolution one week after completion of treatment
        - women with pyelonephritis should be admitted due to risk of premature labour and maternal renal complications

        - if group B Strep is isolated, the antenatal service should be informed so a prophylactic antibiotic can be offered during labour
- influenza
    - consequences
        - may be associated with increased perinatal mortality, prematurity, smaller neonatal size, lower birth weight
        - serious illness from influenza is higher in pregnant women
    - management
        - vaccination with inactivated influenza vaccine (live attenuated vaccine is not recommended)
        - vaccination during pregnancy provides passive immunity to the neonate in the first six months of life
        - early use of antivirals (within 48 hours of symptom onset) is indicated and hospitalisation may be necessary

ObC4 ectopic pregnancy

- anatomy
    - 97% occur in the fallopian tubes
    - as a general rule, only an intrauterine pregnancy is viable
- risk factors
    - 1/3 do not have risk factors
    - history of tubal ligation
    - history of ectopic pregnancy
    - previous tubal infection
    - fallopian and endometrial anomalies
    - fertility treatment
    - endometriosis
    - IUCDs
- clinical features
    - history
        - PV bleeding
        - abdominal/pelvic pain
        - 6-8 weeks LMP
        - shoulder tip pain (large amount of bleeding)
        - lightheaded
        - postural symptoms
    - examination
        - adnexal tenderness and masses
        - state of cervix and material passing through it
        - foetal heart (almost never heard in ectopic)
    - investigations
        - beta-HCG (should almost double every 2 days)
            - if >1200 and there is no intrauterine pregnancy, it is a probable ectopic
        - bloods to investigate for other causes of abdominal pain
        - Rh status
        - MSU

        - TV US
- management
    - surgery
        - unstable
        - large
        - peritonitis
        - surgically managed rhesus-negative patients will require 250IU anti-D
    - methotrexate (single-dose)
        - no peritonitis
        - <3.5cm
        - no free fluid on US
        - ability to closely monitor as an outpatient
        - 75% have abdominal pain 2-3 days after administration; other side effects include nausea, vomiting and reversible impaired liver function
            - contraception should be used for 3-6 months, as methotrexate is teratogenic
    - conservative
        - falling hCG levels and a clinically well patient

ObC5 genital injury/Female Genital Mutilation

- background
    - widely practised throughout Africa and the Middle East
    - WHO definition:
        - 'all procedures that involve partial or total removal of the external female genitalia, or other injury to the female genital organs for non-medical reasons'
    - an estimated 137,000 girls and women in the UK are living with the consequences of FGM and around 60,000 girls under 15 are at risk of FGM
- types
    - type 1 – clitoridectomy
        - partial or total removal of the clitoris and, in very rare cases, only the prepuce
    - type 2 – excision
        - partial or total removal of the clitoris and the labia minor, with or without excision of the labia majora
    - type 3 – infibulation
        - narrowing of the vaginal opening through the creation of a covering seal; the seal is formed by cutting and repositioning the inner or outer labia, with or without removal of the clitoris
    - type 4 – other
        - all other harmful procedures to the female genitalia for non-medical purposes, e.g. pricking, piercing, incising, scraping and cauterising the genital area
- procedure
    - usually carried out between the ages of 5 and 8 but may be done at any time from birth to the delivery of the first baby
    - traditionally carried out by an older woman with no medical training

    - anaesthetics and antiseptics are not generally used
    - it is usually carried out using basic tools such as knives, scissors, scalpels, pieces of glass and razor blades
    - often iodine or a mixture of herbs is placed on the wound to tighten the vagina and stop bleeding
- complications
    - sepsis and death peri-procedure
    - long-term complications:
        - extensive damage of the external reproductive system
        - uterine, vaginal and pelvic infection
        - cysts and neuromas
        - increased risk of vesico-vaginal fistula
        - complications in pregnancy and childbirth
        - psychological damage
        - sexual dysfunction
        - difficulties in menstruation
    - in women with type 3 mutilation, the introitus may be too narrow for childbirth, and the tissues that have sealed together need to be separated (deinfibulation)
- management
    - RCOG recommends that every pregnant woman should be asked if she has been cut
    - all acute NHS hospitals are required to record FGM:
        - if a patient has had FGM
        - if there is a family history of FGM
        - if an FGM-related procedure has been carried out on a woman
    - cases of FGM in girls under 18 should be reported to the police
    - multidisciplinary care is required, including psychological and educational input
    - FGM is an abuse of human rights and a child protection issue

ObC6 HELLP

- background
    - usually presents in women with pre-eclampsia or eclampsia
    - characterised by:
        - **h**aemolysis
        - **e**levated **l**iver enzymes
        - **l**ow **p**latelet count
    - most isolated cases of thrombocytopenia in pregnancy are due to gestational thrombocytopenia rather than HELLP
    - the cause is unknown, but it is a disorder of the placenta
    - there is also an incomplete form with no haemolysis (ELLP)
- risk factors
    - age >35
    - nulliparity
    - previous gestational hypertension
    - multiple pregnancy
    - previous HELLP syndrome
    - Caucasian racial origin
    - antiphospholipid syndrome

- presentation
    - may present at any time in the last half of pregnancy
    - 70% of cases present before delivery
        - peak is between 27 and 37 weeks but may be earlier or later
    - 30% present post-partum, usually within 48 hours of delivery
    - symptoms are usually non-specific
        - malaise
        - fatigue
        - right upper quadrant or epigastric pain
        - nausea
        - vomiting
        - flu-like symptoms
    - onset is usually rapid
    - headache and visual symptoms may occur
    - symptoms are exacerbated at night and relieved during the day
    - hepatomegaly can occur
    - some women have easy bruising/purpura
    - examination may reveal:
        - oedema
        - hypertension
        - proteinuria
        - tenderness over the liver
    - there needs to be a high index of clinical suspicion to avoid diagnostic delay and improve outcome
- investigations
    - haemolysis with fragmented red cells on the blood film, due to microangiopathic haemolytic anaemia
    - raised LDH >600 IU/L with a raised bilirubin, due to destruction of red blood cells
    - liver enzymes are raised with an AST or ALT level of >70 IU/L, due to liver injury
    - platelets <100 $x10^9$/L due to activation and increased consumption
    - levels associated with increased maternal morbidity and mortality are:
        - AST or ALT >150 IU/L
        - LDH >1400 IU/L
        - uric acid >7.8 mg/100ml (>460 μmol/L)
- differential
    - acute fatty liver of pregnancy
    - thrombotic thrombocytopenic purpura
    - immune thrombocytopenia
    - haemolytic uraemic syndrome
    - acute exacerbation of SLE
    - viral hepatitis
    - cholangitis
- management
    - definitive management requires delivery of the foetus, which is advised after 34 weeks of gestation if multisystem disease is present
    - if the foetus is less than 34 weeks and delivery can safely be delayed, corticosteroids should be given to promote foetal lung maturation
    - magnesium sulphate should be considered

    - transfusion of red cells, platelets, fresh frozen plasma and cryoprecipitate or fibrinogen concentrate as indicated clinically and by blood tests
    - postpartum HELLP syndrome may be treated with plasma exchange
    - blood pressure control is essential
    - women with severe liver damage may need liver transplantation
    - women should be screened for antiphospholipid syndrome
- prognosis
    - if not treated early, up to 25% of women may develop serious complications
    - without treatment there is significant mortality
    - the mortality rate among babies varies and depends mainly on gestation and birth weight
- complications
    - maternal
        - eclampsia – 4-9%
        - placental abruption – 9-20%
        - DIC – 5-56%
        - AKI – 7-36%
        - severe ascites – 4-11%
        - cerebral oedema – 1-8%
        - pulmonary oedema – 3-10%
        - wound infection after caesarean – 7-14%
        - liver rupture
        - retinal detachment and other eye problems
        - cerebral haemorrhage or stroke
        - death – 1-25%
    - foetal
        - perinatal death – 7-34% depending on gestational age
        - intrauterine growth restriction – 38-61%
        - preterm delivery – 70%
        - neonatal thrombocytopenia – 15-38%

ObC7 heavy menstrual bleeding

- menorrhagia
    - definition:
        - normal menstrual cycle is blood loss for 7 days with a cycle between 21 and 35 days
        - loss is heaviest for the first few days then tails off
        - average blood loss is 30-40ml, excessive is >80ml and/or duration >7 days
        - NICE definition of heavy menstrual loss is excessive blood loss that interferes with a woman's physical, social, emotional and/or quality of life
    - causes:
        - no cause found in 40-60% (referred to as dysfunctional uterine bleeding)
        - local causes:
            - fibroids
            - endometrial polyps
            - endometriosis
            - adenomyosis

            - endometritis
            - pelvic inflammatory disease
            - endometrial hyperplasia or carcinoma
        - systemic disease:
            - hypothyroidism
            - liver disease
            - kidney disease
            - obesity
            - bleeding disorders (e.g. von Willebrand's)
        - iatrogenic:
            - IUCD
            - anticoagulation
- history:
    - duration of bleeding and how much is heavy
    - length of cycle
    - if tampons and towels worn together (=heavy flow)
    - passage of clots (=heavy flow)
    - associated menstrual problems:
        - premenstrual syndrome
        - intermenstrual bleeding
        - postcoital bleeding
        - dyspareunia
        - pelvic pain
    - contraception and plans for conception
    - symptoms of anaemia
    - effect on personal life and time off work
    - past medical problems including clotting disorders, gynaecological history
    - symptoms of clotting problems (e.g. easy bruising, bleeding gums)
- examination:
    - signs of anaemia
    - abdominal examination
    - body mass index
    - signs of endocrine abnormality
    - consider pelvic examination if indicated
- investigations:
    - pictorial bleeding chart
    - FBC
    - TFTs/other endocrine tests if clinical suspicion of abnormality
    - consider US
- indications for referral to secondary care include:
    - persistent intermenstrual bleeding
    - symptoms not improving with medical management
    - women over 45 with heavy menstrual bleeding
    - history suggestive of endometrial pathology
    - abnormality on physical examination
    - risk factors for endometrial cancer/hyperplasia
    - signs or symptoms suggestive of malignancy

        - surgical treatment wanted by patient
        - iron deficiency anaemia not responding to treatment
    - management:
        - empirical pharmacological treatment (second can be tried if first not working)
            - first line is LNG-IUS-Mirena
                - should be left in for at least 12 months
                - more effective than oral treatment, similar effect to ablation
            - second line
                - tranexamic acid, NSAIDs such as mefenamic acid, combined oral contraceptive pill
                    - mefenamic acid inhibits prostaglandin synthesis, reduces menstrual loss by 25% in ¾ women
                    - tranexamic acid is a plasminogen-activator inhibitor, inhibits dissolution of thrombosis and can reduce flow by up to 50%
                    - COC suppresses production of gonadotrophins and can reduce menstrual loss by up to 50%
            - third line
                - progestogens (norethisterone or injected long-acting progestogens such as Depo-Provera
        - surgical options
            - endometrial ablation
                - removes full thickness of endometrium and retains the uterus
                - contra-indicated if large fibroids or suspected malignancy or family not completed
                - various types of ablation are used and it can be a good alternative to hysterectomy
            - uterine artery embolization or hysteroscopic myomectomy
                - if uterus >10 weeks in size and patient wants to retain uterus
            - hysterectomy
                - with conservation of ovaries if possible
- endometriosis
    - definition:
        - presence of endometrial tissue outside the uterus
            - can have large cystic lesions causing fibrosis and adhesions
    - presentation:
        - severe dysmenorrhoea
        - deep dyspareunia
        - chronic pelvic pain
        - ovulation pain
        - infertility
        - cyclical premenstrual symptoms
    - ED management:
        - NSAIDs

        - exclusion of ectopic
        - outpatient referral to gynaecology
            - diagnosis is by laparoscopy and direct visualisation of pelvis with positive histology
- ovarian cyst rupture
    - presentation:
        - abdominal pain due to peritoneal irritation and localised tenderness
    - management:
        - symptomatic treatment and referral to gynaecology
- ovarian cyst torsion
    - cause:
        - enlarged ovary from cyst or neoplasm torts around the pedicle
        - may resolve spontaneously or progress to ovarian infarction
    - presentation:
        - sudden unilateral lower abdominal pain
        - abdominal and adnexal tenderness
    - management:
        - symptomatic relief
        - referral to gynaecology as inpatient
            - diagnosis by transvaginal ultrasound or laparoscopy
- dysmenorrhoea
    - causes:
        - primary – no underlying pathology
            - worse in first few days of menstruation
            - excess prostaglandins cause painful uterine contractions
            - treatment with prostaglandin inhibitors such as mefenamic acid or NSAIDs
        - secondary – e.g. endometriosis, PID, fibroids
    - management:
        - symptom control, referral to GP
- fibroids
    - definition:
        - nodules of smooth muscle cells and fibrous connective tissue developing within the wall of the uterus
    - symptoms:
        - menorrhagia
        - dysmenorrhoea
        - deep dyspareunia
    - complications:
        - torsion
            - sudden, severe, colicky pain
        - infarction (red degeneration)
            - particularly during pregnancy
    - management:
        - referral to gynaecology
- corpus luteum cyst rupture
    - definition:

        - corpus luteum is formed when a mature ovarian follicle ruptures and releases an ovum
        - rupture of a corpus luteum cyst can cause frank haemorrhage into the peritoneum
    - presentation:
        - abdominal pain and occasionally syncope during second half of menstrual cycle
    - management:
        - may require fluid resuscitation and admission if significant haemorrhage
- mittelschmerz
    - definition:
        - mid cycle pain due to leakage of prostaglandin-containing follicular fluid at ovulation
    - management:
        - symptom control and discharge home

ObC8 hyperemesis gravidarum

- definition:
    - unrelenting, excessive pregnancy-related nausea and/or vomiting that prevents adequate intake or food or fluid
    - rare (1 in 1000)
        - lesser degrees ('morning sickness') are common
- investigations:
    - renal function
        - risk of hyponatraemia, hypokalaemia, renal failure
    - bicarbonate and chloride
        - risk of hypochloraemic metabolic alkalosis
    - FBC
    - LFTs
        - ALT, AST, bilirubin may be increased
    - urine dipstick
        - evidence of UTI
        - increased specific gravity as marker of dehydration
    - ultrasound
        - exclude twins or hydatidiform mole
- management:
    - admit if unable to maintain adequate hydration/nutrition
    - IV fluids
    - thiamine
        - to prevent Wernicke's encephalopathy
    - anti-emetics
        - promethazine and cyclizine have proven safety records
    - LMWH
        - risk of VTE
    - consider PPI
- complications:
    - Mallory-Weiss tear

- Wernicke's encephalopathy
- hyponatraemia
- central pontine myelinosis if sodium corrected too quickly
- B12 and B6 deficiency
- foetal intra-uterine growth retardation
- venous thromboembolism

ObC9 maternal collapse (Green top guideline 56)

- definition:
  - acute event involving the cardiorespiratory and/or central nervous systems resulting in reduced or absent conscious level at any stage in pregnancy and up to 6 weeks after birth
    - may progress to cardiac arrest and death
  - incidence of cardiac arrest in pregnancy is 1 in 36,000 (maternal collapse numbers not known)
  - recognition of deterioration
    - NEWS score can be used up to 20 weeks of gestation
    - use MEOWS (Modified Early Obstetric Warning Score) after this
- causes:
  - vasovagal
  - epileptic seizure
  - haemorrhage
    - major haemorrhage incidence 6 in 1000 pregnancies
    - consider concealed haemorrhage
  - thromboembolism
    - most common cause of direct maternal death
  - amniotic fluid embolism
    - incidence of 1.7 per 100,000 maternities
    - presentation:
      - collapse during labour or birth or within 30 minutes of birth
      - acute hypotension, respiratory distress and acute hypoxia
      - may have seizures and cardiac arrest
      - initial pulmonary hypertension may resolve and progress to left ventricular dysfunction or failure
      - coagulopathy and massive postpartum haemorrhage often occur if the mother survives long enough
    - diagnosis is clinical
  - cardiac disease
    - common, usually with no previous history
    - main causes are ischaemia and sudden arrhythmic cardiac death with a structurally normal heart
    - consider aortic root dissection (and image)
      - central chest or interscapular pain
      - wide pulse pressure (systolic hypertension)
      - new cardiac murmur
    - congenital and rheumatic heart disease numbers are increasing as survival improves

        - other cardiac causes:
            - cardiomyopathy
            - coronary artery dissection
            - acute left ventricular failure
            - infective endocarditis
            - pulmonary oedema
    - sepsis
        - most common organisms in obstetric sepsis are Strep A, B and D, pneumococcus and E. coli
    - drug toxicity and overdose
        - including local anaesthetics and magnesium in maternity care
    - eclampsia
    - intracranial haemorrhage
        - often preceded by headache
        - hypertensive, ruptured aneurysm, AV malformation
    - anaphylaxis
    - other causes
        - hypoglycaemia
        - hyponatraemia
        - other electrolyte and metabolic disturbances
        - airway obstruction (e.g. secondary to aspiration or foreign body
        - air embolism
        - tension pneumothorax
        - cardiac tamponade secondary to trauma or dissection
        - hypothermia
- changes in pregnancy that affect resuscitation:
    - aortocaval compression
        - venous return reduced from about 20 weeks onwards
        - cardiac output reduced by 30-40%
        - CPR is less effective
        - left lateral uterine displacement required
    - respiratory changes
        - increased tidal volume
        - splinting of diaphragm by enlarged uterus
            - reduced functional residual capacity
            - more difficult ventilation
            - patients become hypoxic much more rapidly when hypoventilation occurs
    - intubation
        - likely to be difficult due to weight gain, large breasts and laryngeal oedema
    - aspiration
        - increased risk
            - progesterone relaxes lower oesophageal sphincter
            - uterus raises intra-abdominal pressure
            - delayed gastric emptying during labour or after opioids
            - aspiration pneumonia can be severe (Mendelsson's syndrome)
    - circulation

        - increased cardiac output and hyperdynamic circulation
            - large volumes of blood can be lost rapidly
        - uterus receives 10% of maternal cardiac output at term
            - can have dramatic blood loss
        - up to 35% of circulation can be lost before becoming symptomatic
            - tachycardia may be the only early sign
            - blood loss is tolerated less well if there is significant anaemia
- modifications to resuscitation (from 20 weeks)
    - emergency call for obstetric, obstetric anaesthetic and neonatal (if >22 weeks) teams
    - manual displacement of uterus to left
        - up, off and over
        - or left lateral tilt to 15-30° on firm surface
    - immediate intubation with cuffed ET tube by experienced anaesthetist
        - with plan for failed intubation
    - chest compressions and IV access as per usual resuscitation
        - IV access ideally above diaphragm
    - aggressive fluid replacement (caution in pre-eclampsia/eclampsia)
    - consider US for concealed haemorrhage
    - same defibrillation energies
        - consider AP pad placement if breasts large/engorged
    - no change to ALS drugs
    - perimortem c-section in women >20 weeks gestation if no response to CPR after 4 minutes to assist maternal resuscitation
        - if successful, prompt transfer to appropriate environment
            - expect haemorrhage once circulation restored
    - in collapse secondary to antepartum haemorrhage the foetus and placenta should be delivered to allow control of bleeding
    - IV tranexamic acid significantly reduces mortality in postpartum haemorrhage
    - management of amniotic fluid embolism is supportive
        - early aggressive treatment of coagulopathy including FFPs

ObC10 post-partum haemorrhage

- definition:
    - primary post-partum haemorrhage
        - blood loss of >500ml in first 24h post-delivery
        - can be minor (500-1000ml) or major (>1000ml)
    - secondary post-partum haemorrhage
        - excessive blood loss from the genital tract between 24 hours and 12 weeks post-natally
- risk factors:
    - placental abruption
    - placenta praevia
    - placenta accreta (abnormally adherent placenta)
    - multiple pregnancy
    - pre-eclampsia/gestational hypertension
    - previous post-partum haemorrhage

    - haemophilia
    - anti-coagulant use
- causes:
    - primary post-partum haemorrhage
        - uterine atony (commonest cause)
        - retained placenta
        - genital tract trauma
            - vaginal or cervical lacerations, uterine rupture
        - DIC
    - secondary post-partum haemorrhage
        - retained products of conception
        - intrauterine infection
        - genital tract trauma
        - trophoblastic disease
        - DIC
- management (primary post-partum haemorrhage):
    - immediate activation of obstetric team
    - manage in resus
    - assess airway and breathing, start high-flow oxygen
    - two large bore cannulae
        - send blood for FBC, clotting, fibrinogen
        - urgent cross match of at least 4 units
            - make transfusion department aware massive transfusion may be needed
    - fluid resuscitation
        - initially crystalloid, then cross-matched blood when available
        - consider type specific or type O if cross-matched not available in time
        - may need blood products such as platelets, FFPs, cryoprecipitate
    - stimulation of uterine contraction
        - bi-manual uterine compression (rubbing up the fundus)
        - pharmacologically with oxytocin and ergometrine IV
    - may need surgical control of bleeding
- management (secondary post-partum haemorrhage):
    - manage airway and breathing as needed
    - fluid resuscitation as needed
    - IV antibiotics (e.g. ampicillin and metronidazole) if infection suspected
        - send IV antibiotics and low vaginal swab prior to giving antibiotics
    - refer to obstetrics

ObC11 pre-eclampsia/eclampsia

- definition (pre-eclampsia):
    - three main features:
        - hypertension (systolic >140mmHg or diastolic >90mmHg, or a rise above booking BP of systolic >30mmHg or diastolic >15mmHg)
        - proteinuria (>0.3g/24h)
        - oedema
    - onset usually after 20 weeks gestation

    - affects around 6% of pregnancies
    - eclampsia occurs in 1-2% of pre-eclamptic patients
        - when convulsions occur
- risk factors:
    - nulliparity
    - multiple pregnancy
    - hydatidiform mole
    - age <20 or >40
    - previous pre-eclampsia
    - pre-existing hypertension
    - diabetes or insulin resistance
    - renal disease
    - high body mass index
    - antiphospholipid syndrome
    - long interval between pregnancies (>10 years)
- clinical features:
    - malaise
    - right upper quadrant and epigastric pain
        - due to liver oedema and haemorrhage
    - headache, visual disturbance and papilloedema
        - due to cerebral oedema
    - occipital lobe blindness
    - hyperreflexia, clonus
    - convulsions
        - due to cerebral oedema
        - onset of eclampsia
- investigations:
    - FBC
        - thrombocytopaenia
        - haemoconcentration
    - blood film
        - may develop microangiopathic haemolytic anaemia
    - clotting screen
        - check if thrombocytopaenic
    - renal function
        - risk of renal failure
    - LFTs
        - elevated in HELLP syndrome
    - urine dipstick
        - ≥2+ protein indicates significant proteinuria and need for 24h collection
    - foetal monitoring
        - ultrasound and cardiotocography
- management (pre-eclampsia):
    - early obstetrician referral
    - management in resus
    - consider left lateral position
    - control hypertension with IV labetalol or hydralazine
    - careful fluid management

        - maternal death can occur due to pulmonary oedema with fluid overload
        - limit to around 1ml/kg/h
        - monitor urine output
    - consider magnesium in women with severe pre-eclampsia (systolic ≥170mmHg or diastolic ≥110mgHg plus significant proteinuria - >1g/L)
    - delivery as definitive treatment
        - 44% of eclampsia occurs post-partum
- management (eclampsia):
    - assess airway and breathing adequacy
    - high flow oxygen
    - early consideration of intubation
        - increased aspiration risk and ventilatory inadequacy
    - magnesium to control seizures
        - 4g IV loading dose over 5-10 minutes
        - then maintenance of 1g/h for 24 hours
        - further 2g bolus can be given if recurrent seizures
- complications:
    - CNS
        - intra-cerebral haemorrhage
        - cerebral oedema
        - cortical blindness
        - retinal oedema and blindness
    - liver
        - HELLP – haemolysis, elevated liver enzymes and low platelets
        - intra-abdominal haemorrhage (hepatic rupture)
        - jaundice
    - respiratory
        - pulmonary oedema
    - cardiac
        - cardiac failure
    - renal
        - renal cortical necrosis
        - renal tubular necrosis
    - coagulation
        - DIC
        - microangiopathic haemolysis
        - venous thromboembolism
    - placental
        - placental abruption
        - placental infarction
    - foetal
        - intra-uterine growth retardation
        - premature delivery
        - perinatal death (15% in eclampsia)

ObC12 pelvic infection

- definition:

    - pelvic inflammatory disease is the result of infection ascending from the endocervix causing endometritis, salpingitis, oophoritis, tubo-ovarian abscess and/or peritonitis
- causes:
    - sexually transmitted (90%)
        - Chlamydia
        - Gonorrhoea
        - Mycoplasma genitalium
    - non-sexually transmitted (10%, often post-instrumentation)
        - E. coli
        - Group B strep
        - Bacteriodes
        - Gardenella
- clinical features:
    - lower abdominal pain and tenderness
    - abnormal vaginal or cervical discharge
    - fever (>38°)
    - abnormal vaginal bleeding
        - intermenstrual, post-coital or breakthrough
    - deep dyspareunia
    - cervical excitation
    - adnexal tenderness +/- mass
- investigations:
    - endocervical swab for Chlamydia and Gonorrhoea
    - urinary pregnancy test
    - bloods (ESR, CRP, WCC can be helpful but not specific)
    - transvaginal ultrasound
        - may show inflamed/dilated Fallopian tubes or abscess
- management:
    - empirical treatment
        - low threshold recommended because of lack of definitive clinical diagnostic criteria and potential consequences of not treating
        - mild to moderate
            - oral ofloxacin 400mg BD and metronidazole 400mg BD for 14 days
    - inpatient management:
        - indications:
            - clinically severe disease
            - tubo-ovarian abscess
            - PID in pregnancy
            - intolerance or lack of response to oral therapy
            - surgical emergency not excluded
        - IV ceftriaxone and doxycycline
        - surgical drainage may be needed for tubo-ovarian abscess
        - consider removal of IUCD, especially if symptoms not resolving within 72 hours
    - contact tracing:
        - sexual partners from previous 6 months should be contacted and offered screening via GUM clinic

- complications:
    - infertility
    - ectopic pregnancy (5x increased risk)
    - chronic pelvic pain
    - peritonitis
    - abscess formation

ObC13 post menopausal bleeding

- causes:
    - atrophic vaginitis
    - endometrial polyps
    - fibroids
    - endometrial hyperplasia
    - endometrial carcinoma
    - cervical carcinoma
    - vaginal carcinoma
    - bleeding from non-gynaecological sites (e.g urethra, bladder, lower GI tract)
- investigation:
    - abdominal examination
    - speculum examination
    - bimanual vaginal examination
- management:
    - outpatient referral for gynaecology for further investigation

ObC14 prescribing in pregnancy

- background
    - teratogenicity is the risk of structural abnormality in the first trimester
    - fetotoxicity is the potential functional damage that can occur with exposure to medication in later pregnancy
    - however, maternal illness may also be detrimental to the foetus and women may require medication
    - some drugs have a more robust safety profile in pregnancy
        - few large scale randomised controlled trials have been carried out on pregnant women
    - 50% of pregnant women will take a drug at some point in their pregnancy
    - non-compliance can be an issue as women are reluctant to take medications, and some may have spontaneously stopped – concerns must be addressed
    - if unsure, the doctor should seek advice
- teratogenic drugs
    - may be associated with structural abnormalities in the developing foetus
    - greatest risk is from 5-10 weeks of gestation
    - the possibility of a woman of childbearing age conceiving whilst taking a medication should be considered when prescribing
    - the risk of major malformation in the general population is 2%, so any drug being taken will not necessarily be responsible for a major malformation

    - most women will not present until at least 6 weeks, by which time much of the structural development has occurred, so suddenly stopping or changing drugs may be unnecesary
- fetotoxicity
    - can occur at any time between the late first trimester and birth
    - an example is NSAIDs, which can cause foetal renal dysfunction in the second and third trimester and premature closure of the ductus arteriosus in the third trimester
- priorities and principles
    - the aim is to balance the risks of the potential adverse effects of the drugs (usually to the foetus) with the benefit (usually to the mother) of treating the disease
    - there are three reasons why a woman takes medication during pregnancy:
- inadvertent exposure to medication
- intended continuation of treatment for a pre-existing condition
- initiation of treatment in pregnancy – for example for infection or for a pregnancy-related problem such as pre-eclampsia
- management principles
    - inadvertent exposure
        - obtain accurate details of exposure and gestational age
        - check for confounding family and personal medical history
        - obtain up to date information about published risks of the drug in humans
        - emphasise background risk in counselling
        - be clear what is known (absence of data does not equal no risk)
    - intended continuation or initiation
        - medication should only be used if the intended benefits outweigh the risks
        - try to avoid first trimester use
        - use drugs that have been used extensively in pregnancy, not new ones
        - use the minimum dose required to achieve the desired effect
        - absence of data does not imply safety
- sources of information
    - British National Formulary
    - UK Teratology Information Service
    - Organisation of Teratology Information Specialists
    - Food and Drug Administration
- cardiovascular disease
    - pregnancy
        - avoid ACEi – teratogenic in first trimester; risk of foetal renal dysfunction, oligohydramnios and foetal growth restriction in the second and third trimester
        - methyldopa has a good safety record as an antihypertensive in pregnancy
        - labetolol is licensed for use in pregnancy; there is uncertainty about other beta blockers
        - nifedipine is used widely in obstetric practice (UK guidelines advise only after 20 weeks); modified or slow release preparations should be used
        - diuretics are generally avoided, but low dose thiazide diuretics may be used in the second and third trimester if there is a specific indication
        - hydralazine is often used parenterally in severe hypertension; it is safe for the foetus but has a significant side effect profile

    - breastfeeding
        - beta blockers are considered safe, but the infant should be monitored for signs of beta blockade if maternal dose is high
        - methyldopa is safe but has a potential side effect of low mood so may be best avoided post partum
        - nifedipine is likely to be safe but data is lacking
        - furosemide and thiazide diuretics are probably safe in small to moderate doses
        - captopril and enalapril are probably safe
        - amiodarone should be avoided – long half life and risk of neonatal hypothyroidism
- pro-thrombotic conditions
    - pregnancy
        - warfarin crosses the placenta, is teratogenic and can cause foetal bleeding
        - low dose aspirin (75mg OD) is safe; analgesic doses are contraindicated, particularly in the third trimester because of the risk of premature closure of the ductus arteriosus
        - LMWH and unfractionated heparin do not cross the placenta
    - breastfeeding
        - UFH and LMWH are not excreted in breast milk and are safe
        - warfarin is not excreted in breast milk
        - avoid other oral anticoagulants
        - low dose aspirin in safe
- asthma
    - pregnancy
        - steroids and beta 2 agonist inhalers are safe
        - aminophylline is probably safe and advocated for use in pregnancy
        - oral steroids should not be withheld in acute severe asthma
    - breastfeeding
        - salbutamol, terbutaline and salmeterol inhalers are safe
        - theophylline may cause toxicity in younger infants and a slow release preparation should be used
        - inhaled steroids are safe and oral corticosteroids are considered safe
- epilepsy
    - pregnancy
        - indication for medication should be reviewed
            - it may be appropriate to consider dose reduction or withdrawal (e.g. childhood epilepsy and seizure free for many years)
            - however this will necessitate stopping driving
            - main risk factors for recurrence are presence of tonic-clonic and myoclonic seizures and initial difficulty controlling the epilepsy
        - all anti-epileptic drugs are known to increase the risk of congenital malformations, but the majority of women taking them give birth to healthy babies
        - 5mg folic acid should be started at least 6 weeks before conception and continued for at least the first trimester
        - monotherapy is less teratogenic than polytherapy

        - sodium valproate is considered particularly teratogenic
        - lamotrigine levels fall by 50% from the first trimester, often with accompanying worsening of seizures – a pre-emptive increase in the second trimester should be considered
        - poorly controlled epilepsy is potentially dangerous for mother and foetus
    - breastfeeding
        - phenytoin, carbamazepine and sodium valproate are excreted in breast milk, but considered safe if drug levels in the mother are kept within therapeutic range
        - breastfeeding should be avoided if taking phenobarbitone, as it can accumulate in the infant, resulting in sedation; withdrawal effects are also possible
        - lamotrigine may accumulate because of the infant's immature metabolism
        - there is no information about the effects of the newer epileptic drugs
- depression
    - pregnancy
        - mild-moderate depression should be managed with psychological therapy alone if possible
        - when medication is deemed necessary, a tricyclic antidepressant should be considered
        - SSRIs are relatively new drugs, with fluoxetine being the SSRI of choice in pregnancy
    - breastfeeding
        - tricyclic antidepressants are probably safe
        - sertraline is the SSRI of choice
- thyroid disease
    - pregnancy
        - hypothyroidism – most women require an increase in their thyroxine dose; adequate intake of iodine is also important
        - hyperthyroidism – avoid radioisotopes; propylthiouracil is preferred to carbimazole
    - breastfeeding
        - thyroxine is safe and should be continued
        - theoretical risk of thyroid suppression with carbimazole – neonatal thyroid function tests should be monitored
        - propylthiouracil is probably safe – infant thyroid function should be monitored
        - radioactive iodine is contraindicated – can cause permanent thyroid damage in the infant
- diabetes
    - pregnancy
        - establish tight glycaemic control before conception
        - if type 2 or previous gestational diabetes, encourage weight loss before conception
        - start folic acid 5mg/day before conception
        - insulin requirement increases during pregnancy
    - breastfeeding
        - insulin is safe because it does not pass into breast milk

        - metformin is probably safe
- rheumatic disease
    - pregnancy
        - disease activity must be controlled to avoid adverse effects due to disease flare
        - analgesia – paracetamol is safe; avoid NSAIDs, particularly in the third trimester
        - prednisolone is safe (caution with high doses, implicated in PROM)
        - hydroxychloroquine and sulfasalazine are the safest immunosuppressant options
        - azathioprine is probably safe
        - methotrexate and cyclophosphamide are contraindicated because of their teratogenic potential
        - biologics appear to be safe (but there is limited data) in the first trimester – there is no transplacental transfer until around 16 weeks; transplacental transfer increases toward term
        - associated medications should be reviewed
    - breastfeeding
        - paracetamol is safe
        - NSAIDs are safe but COX-2 selective inhibitors should be avoided; analgesic doses of aspirin should be avoided because of the risk of platelet dysfunction and Reye's syndrome
        - mesalazine and sulfasalazine can cause diarrhoea, which resolves on stopping the drug
        - prednisolone is excreted into breast milk but in small quantities; there are no reports of serious adverse effects and it is considered safe
        - azathioprine is probably safe
- antibiotics
    - pregnancy
        - full adult dose and length of treatment should be given
        - serious infections should be treated aggressively
        - penicillins and cephalosporins are considered safe – higher dose regimens should be used as they are cleared more quickly due to increase in GFR during pregnancy
        - aminoglycosides should be avoided for minor infections because of the theoretical risk of neonatal ototoxicity seen with streptomycin
        - avoid tetracyclines – they cause discolouration and dysplasia of bones and teeth
        - nitrofurantoin is safe except near term as risk of neonatal haemolytic anaemia
        - chloramphenicol is safe topically but other routes of administration are associated with grey baby syndrome
    - breastfeeding
        - may cause altered gut flora and thus diarrhoea
        - if the infant develops a fever, take into account maternal antibiotic treatment
        - penicillins, cephalosporins and erythromycin are safe
        - sulfonamides have a risk of kernicterus in preterm, ill or stressed infants

        - metronidazole may give breast milk an unpleasant taste
        - chloramphenicol should be avoided due to risk of bone marrow suppression

ObC15 Rhesus D prophylaxis

- pathophysiology:
    - a rhesus negative mother exposed to blood of rhesus positive foetus may develop anti-D antibodies
    - antibodies are IgG and not usually a problem in initial pregnancy because they do not have time to develop before delivery
    - in subsequent pregnancy with rhesus positive foetus, antibodies are formed and can cross the placenta
    - the antibodies attack and destroy red blood cells of the foetus causing haemolytic disease of the newborn
- sensitising episodes before delivery:
    - closed abdominal injury (particularly in 3rd trimester)
    - antepartum haemorrhage
    - intrauterine death
    - invasive prenatal diagnostics (amniocentesis, chorionic villus sampling)
    - ectopic pregnancy
    - spontaneous miscarriage
        - all pregnancies >12/40
        - <12/40 if intervention required to evacuate the uterus
    - threatened miscarriage
        - all pregnancies >12/40
        - <12/40 if heavy or repeated bleeding, or associated with abdominal pain
    - therapeutic termination
- anti-D
    - recommended for sensitising episodes
    - routine antenatal prophylaxis:
        - 500 IU at 28 weeks and 34 weeks gestation
    - prophylactic administration following a sensitising episode:
        - 250 IU up to 20/40 gestation
        - 500 IU after 20/40 gestation, plus Kleihauer test to detect large foeto-maternal haemorrhage and direct further doses as required
    - following delivery:
        - 500 IU, plus Kleihauer test
    - given IM, and should be as soon as possible
        - must be within 72 hours of sensitising episode

ObC16 sepsis in and following pregnancy

- sepsis in pregnancy (Green-top guideline 64a)
    - background
        - sepsis in pregnancy remains an important cause of maternal death in the UK
        - definition: infection plus systemic manifestations of infection; severe sepsis is sepsis plus sepsis-induced organ dysfunction or tissue hypoperfusion; septic shock is the persistence of hypoperfusion despite adequate fluid replacement therapy

        - urinary tract infection and chorioamnionitis are common infections associated with septic shock in pregnant patients
        - a MEOWS chart should be used for recording observations and a high index of suspicion is required as signs and symptoms may be less distinctive
    - risk factors for maternal sepsis
        - obesity
        - impaired glucose tolerance/diabetes
        - impaired immunity/immunosuppressant medication
        - anaemia
        - vaginal discharge
        - history of pelvic infection
        - history of group B streptococcal infection
        - amniocentesis and other invasive procedures
        - cervical cerclage
        - prolonged spontaneous rupture of membranes
        - GAS infection in close contacts/family members
        - black and other minority ethnic group origin
- clinical features suggestive of sepsis
    - fever or rigors
    - diarrhoea or vomiting – may indicate exotoxin production
    - rash
    - abdominal/pelvic pain and tenderness
    - offensive vaginal discharge
    - productive cough
    - urinary symptoms
- diagnostic criteria for sepsis in pregnant women in presence of documented or suspected infection
    - general
        - fever >38°
        - hypothermia <36°
        - tachycardia >100
        - tachypnoea >20
        - impaired mental state
        - significant oedema or positive fluid balance (>20ml/kg over 24 hours)
        - hyperglycaemia in the absence of diabetes (glucose >7.7 mmol/L)
    - inflammatory variables
        - WCC >12 (note that a transient leucocytosis is common in labour)
        - leucopenia (WCC <4)
        - normal WCC with >10% immature forms
        - CRP >7
    - haemodynamic variables
        - arterial hypotension (systolic <90; MAP <70; systolic decrease >40mmHg)
    - tissue perfusion variables
        - raised lactate ≥4
        - decreased capillary refill or mottling
    - organ dysfunction variables
        - arterial hypoxaemia (PaO_2/FiO_2 <40kPa) – severe if <33.3kPa in the absence of pneumonia or <26.7kPa in the presence of pneumonia

        - oliguria (<0.5ml/kg/hr for at least 2 hours, despite adequate fluid resuscitation)
        - creatinine rise of >44.2μmol/L
        - coagulation abnormalities (INR >1.5 or APTT >60s)
        - thrombocytopaenia (<100)
        - hyperbilirubinaemia (>70)
        - ileus (absent bowel sounds)
- staphylococcal toxic shock (5/5 = confirmed, 4/5/ = probable)
- fever ≥39.9°
- rash – diffuse macular erythroderma
- desquamation – 10 to 14 days after onset of illness, especially palms and soles
- hypotension – systolic <90
- multisystem involvement
        - three or more of:
            - gastrointestinal – vomiting or diarrhoea at onset of illness
            - muscular – severe myalgia or elevated CK
            - mucous membranes - vaginal, oro-pharyngeal or conjunctival hyperaemia
            - renal – creatinine twice the upper limit of normal
            - hepatic - total bilirubin twice the upper limit of normal
            - haematological - platelets ≤100
            - CNS – disorientation or alterations in consciousness without focal neurological signs
- streptococcal toxic shock syndrome (definite: meets clinical case definition plus isolation of group A strep from a normally sterile site; probable: meets clinical case definition plus isolation from non-sterile site)
    - A – isolation of beta-haemolytic group A Streptococcus from:
        - normal sterile site – blood, CSF, peritoneal fluid, tissue biopsy
        - non-sterile site – throat, vagina, sputum
    - B – clinical case definition
        - multi-organ involvement characterised by:
- hypotension PLUS
- two or more of:
            - renal impairment – creatinine >176
            - coagulopathy – platelets <100 or DIC
            - liver involvement – ALT or aspartame
            - acute respiratory distress syndrome
            - generalised erythematous macular rash (present in 10%) – may desquamate
            - soft tissue necrosis including necrotising fasciitis, myositis or gangrene
- indications for transfer to ICU
    - cardiovascular
        - hypotension or raised lactate persisting despite fluid resuscitation, suggesting the need for inotrope support
    - respiratory
        - pulmonary oedema

        - mechanical ventilation
        - airway protection
    - renal
        - renal dialysis
    - neurological
        - significantly decreased conscious level
    - miscellaneous
        - multi-organ failure
        - uncorrected acidosis
        - hypothermia
- antimicrobial choice
    - should be broad spectrum and as per local guidance
    - limitations of antimicrobials include:
        - co-amoxiclav
            - does not cover MRSA or Pseudomonas
            - there is concern about an increase in the risk of necrotising enterocolitis in neonates exposed to co-amoxiclav in utero
        - metronidazole
            - only covers anaerobes
        - clindamycin
            - covers most streptococci and staphylococci, including many MRSA, and switches off exotoxin production with significantly decreased mortality
            - not renally excreted or nephrotoxic
        - piperacillin-tazobactam and carbapenems
            - covers all except MRSA and are renal sparing
        - gentamicin (single dose 3-5mg/kg)
            - poses no problem in normal renal function
            - serum levels must be monitored if doses to be given regularly
- IVIG
    - recommended for severe invasive streptococcal or staphylococcal infection if other therapies have failed
- foetal monitoring and delivery
    - delivery should be considered in a critically unwell woman if it would be beneficial to mother, baby or both
    - if preterm delivery is anticipated, consideration of corticosteroids should be made
    - continuous electronic foetal monitoring in the intrapartum period
    - epidural/spinal anaesthesia should be avoided in women with sepsis and a general anaesthetic will usually be required for c-section
- prophylaxis
    - neonates of mothers with invasive group A strep in the peripartum period require prophylactic antibiotics
    - close household contacts should be warned to seek medical attention if symptoms develop and may require antibiotic prophylaxis
    - healthcare workers exposed to respiratory secretions should be considered for antibiotic prophylaxis
- sepsis following pregnancy (Green-top guideline 64b)

    - background
        - there has been a rise in maternal deaths attributable to group A beta-haemolytic streptococci
        - most common site of sepsis in the puerperium (within 6 weeks of giving birth) is the genital tract and uterus, resulting in endometritis
        - disease progression can be rapid; patients with constant severe abdominal pain and tenderness unrelieved by analgesia should have urgent review
- common organisms in puerperal sepsis
    - GAS (Streptococcus pyogenes)
    - Escherichia coli
    - Staphylococcus aureus
    - Streptococcus pneumoniae
    - MRSA, Clostridium septicum, Morganella morganii
- specific infections
    - mastitis
        - may lead to breast abscesses, necrotising fasciitis and toxic shock syndrome
        - warning signs include being clinically unwell, no response to oral antibiotics in 48 hours, recurrence or very severe/unusual symptoms
    - urinary tract infection
        - acute pyelonephritis should be treated aggressively
        - women with signs of sepsis, unable to remain hydrated or with vomiting should be admitted
    - skin and soft tissue
        - consider cannula and injection sites and episiotomy sites
        - indwelling lines should be removed as soon as possible if they may be a source of infection
        - septicaemic seeding from a uterine focus can give rise to a secondary focus in a limb, resembling a venous thrombosis
    - infection related to regional anaesthesia
        - spinal abscess is a very rare complication
        - the diagnosis should be considered and investigated as permanent spinal cord or cauda equina may occur if neural compression is prolonged
- common symptoms of sepsis in the puerperium
    - fever, rigors (beware normal temperature after antipyretics)
    - diarrhoea or vomiting (may indicate exotoxin production)
    - breast engorgement/redness
    - rash (generalised maculopapular)
    - abdominal/pelvic pain and tenderness
    - wound infection – spreading cellulitis or discharge
    - offensive vaginal discharge
    - productive cough
    - urinary symptoms
    - delay in uterine involution, heavy lochia
    - general, non-specific signs such as lethargy, reduced appetite
- red flag signs and symptoms
    - pyrexia >38°
    - sustained tachycardia >90
    - breathlessness (RR >20)

    - abdominal or chest pain
    - diarrhoea and/or vomiting
    - uterine or renal angle pain and tenderness
    - woman is generally unwell or seems unduly anxious or distressed
- management
    - broad spectrum antibiotics – should be discussed with microbiologist if breastfeeding
    - IVIG in severe sepsis
    - PPE worn by those caring for the patient

ObC17 thrombosis during and following pregnancy (Green-top guideline 37b)

- background
    - risk of VTE in pregnant women is 4-5 fold higher than in non-pregnant women of the same age
        - absolute risk is low, around 1 in 1000 pregnancies
    - the puerperium is the time of highest risk
- summary of guideline
    - any woman with signs or symptoms suggestive of VTE should have objective testing and treatment with LMWH until diagnosis excluded (unless contraindicated) – hospitals should have an agreed protocol
    - DVT investigation
        - compression duplex US should be undertaken when there is clinical suspicion of DVT
            - if US is negative with low level of clinical suspicion, anticoagulation can be discontinued
            - if US is negative with high level of clinical suspicion, anticoagulation should be discontinued and the US repeated on days 3 and 7
    - PE investigation
        - ECG and CXR should be performed
            - abnormal features caused by PE include:
                - atelectasis
                - effusion
                - focal opacities
                - regional oligaemia
                - pulmonary oedema
        - if there are also signs and symptoms of DVT, compression duplex US should be performed
            - if DVT is confirmed, treatment for VTE should continue, with no further investigation
        - if there are no signs/symptoms of DVT a V/Q scan or CTPA should be performed
            - if CXR is abnormal and there is clinical suspicion of PE, CTPA should be performed in preference to V/Q scan
        - if V/Q scan or CTPA is normal but clinical suspicion of PE remains, alternative or repeat testing should be carried out with anticoagulant treatment continued until PE definitively ruled out

        - CTPA can also identify other pathology including pneumonia, pulmonary oedema and occasionally aortic dissection
        - women should be advised that V/Q scanning may carry a slightly increased risk of childhood cancer compared to CTPA but a lower risk of maternal breast cancer
            - radiation dose to breast tissue in CTPA depends on breast size, technique used and age of woman (risk is higher in younger women)
            - the absolute risk is very small in both
            - women should be involved in the decision where possible
    - other investigation
        - d-dimer testing should not be performed in pregnancy
        - there is no role for pretest probability assessment
        - baseline bloods
            - before starting anticoagulation, FBC, coagulation, U&Es and LFTs should be tested
    - management
        - LMWH should be given in doses titrated against the woman's booking or early pregnancy weight
        - recommended dose is 1.5mg/kg enoxaparin OD
    - massive life-threatening PE in pregnancy or puerperium
        - shocked patients should have a team assessment including a consultant obstetrician
        - women should be managed on an individual basis regarding IV unfractionated heparin, thrombolytic therapy or thoracotomy and surgical embolectomy
        - IV unfractionated heparin is the preferred initial treatment in massive PE with cardiovascular compromise
            - loading dose of 80 units/kg followed by continuous IV infusion of 18 units/kg/hr
            - if thrombolysis was given the loading dose should be omitted
            - APTT must be measured 4-6 hours after the loading dose, 6 hours after any dose change and at least daily when in the therapeutic range
        - urgent portable echo or CTPA within 1 hour is advised and immediate thrombolysis considered if massive PE confirmed
    - additional therapies
        - in initial management of DVT, the leg should be elevated and a graduated elastic compression stocking applied to reduce oedema
        - temporary IVC filters should be considered in the peripartum period for patients with iliac vein VTE to reduce risk of DVT, or in patients with proven DVT and recurrent PE despite adequate anticoagulation
    - maintenance treatment of DVT/PE
        - therapeutic LMWH should be continued through the rest of the pregnancy and for at least 6 weeks postnatally and until at least 3 months of treatment has been given in total
        - vitamin K antagonists such as warfarin should not be used antenatally due to adverse effects on the foetus

        - consideration should be given to the newer oral anticoagulants in women unable to tolerate heparin
        - post delivery
            - after 3 months of treatment, continuing risk of thrombosis should be assessed
            - women should be offered LMWH or oral anticoagulation – with a discussion about regular blood tests required for warfarin, particularly in the first 10 days
            - postpartum warfarin should be avoided until at least the fifth day and for longer in women at increased risk of postpartum haemorrhage
            - neither heparin nor warfarin is contraindicated in breastfeeding
    - labour and delivery
        - no further LMWH should be injected once the woman is in established labour or thinks that she is in labour
        - when delivery is planned, LMWH should be stopped 24 hours prior
        - regional anaesthetic or analgesic procedures should not be carried out until at least 24 hours after the last LMWH dose
        - LMWH should not be given for 4 hours after the use of spinal anaesthesia or after the epidural catheter has been removed

ObC18 trauma in pregnancy

- background
    - optimal management of the mother increases survival chances for the foetus
- anatomical considerations
    - the enlarged uterus is more prone to injury once it is outside the pelvis (>12/40)
        - also makes abdominal assessment difficult
    - bony pelvis is less prone to fracture but retroperitoneal haemorrhage may be massive due to increased vascularity
    - IVC compression when supine causing hypotension and potential for increased bleeding from lower limbs due to increased venous pressure
        - decompress with manual displacement of the uterus or use of a wedge to achieve left lateral position
    - higher diaphragm results in decreased residual volume
    - airway is difficult to control
            - large breasts
            - full dentition
            - neck oedema
            - obesity
        - to facilitate intubation, consider:
            - laryngoscope with short or tilted handle
            - bougie
            - video laryngoscopy
        - cricothyroidotomy may be more difficult due to soft tissue changes
        - cricoid pressure should be used and the stomach decompressed early to reduce risk of aspiration
    - breathing

        - oxygenation
            - high flow O2 should be administered until hypoxaemia, hypovolaemia and foetal distress are excluded
            - FRC is decreased because of increased intra-abdominal pressure; which predisposes to rapid desaturation
        - ventilation
            - increased tidal volumes due to progesterone means hyperventilation is normal and bicarbonate is normally low reflecting a compensated respiratory alkalosis
            - there is decreased thoracic compliance due to breast enlargement and increased intra-abdominal pressure
            - bag valve mask ventilation is more difficult
        - chest drains
            - should be placed higher (3rd or 4th intercostal space) as diaphragms may be higher
    - circulation
        - vital signs are altered in pregnancy, mimicking shock
        - heart rate increases by 10-15bpm by the third trimester
        - BP is 10-15mmHg lower by the second trimester then increases to near normal by term
        - cardiac output increases by 1-1.5 L/min by the end of the first trimester due to increased blood volume and decreased systemic vascular resistance
        - CVP is lower due to decreased venous return
        - fluid resuscitation
            - uterine blood flow is not autoregulated, so hyper-hydration is preferable as maternal compensation for blood loss will be at the expense of the foetus
            - women with pre-eclampsia in particular are more prone to fluid overload
        - hypotension and shock
            - supine hypotension syndrome from compression of the IVC by the uterus may occur
                - may decrease cardiac output by 30%
                - after 24 weeks, left lateral tilt should be used
            - evidence of hypovolaemia may not be apparent until about 1500 ml of blood has been lost due to increase in blood volume during pregnancy of around 50%
        - pituitary is twice normal size and at risk of infarction if hypovolaemia occurs (Sheehan's syndrome)
    - abdominal and GI
        - predisposition to regurgitation and vomiting
            - decreased gastric emptying rate due to progesterone
            - increased intra-abdominal pressure
        - early decompression with NG tube should be considered
        - intra-abdominal organs are displaced by the enlarged uterus
        - the intestines move cephalad and are relatively protected by the uterus
        - the position of the liver and spleen is largely unchanged

        - the bladder is an intra-abdominal organ after the first trimester
        - injury to the uterus or vessels may lead to severe bleeding – there is around 1 L/min blood flow to the uterus late in pregnancy
    - haematological
        - relative anaemia is normal due to increased blood volume
        - leucocytosis is a normal feature
        - there is a normal coagulation profile except for elevated fibrinogen and d-dimer
        - blood transfusion need to consider Rh status, and anti-D should be given to Rh- women
        - autotransfusion may occur after delivery due to sequestration of blood in the placenta
    - musculoskeletal
        - the pelvis is different
            - interpretation of pelvic x-rays needs to account for widening of the pubic symphysis and sacroiliac joints by the 7th month
            - pelvic fractures can be associated with uterine injury or massive retroperitoneal haemorrhage due to injury of enlarged uterine vessels
            - pelvic binders may not fit pregnant women
- physiological considerations:
    - pregnant patients can tolerate up to 35% blood loss before showing signs of hypovolaemia
        - foetus may be compromised before this
    - functional residual capacity decreased and oxygen requirements increased so hypoxia develops more quickly
    - increased risk of aspiration
        - decreased oesophageal pressure
        - increased gastric pressure
        - prolonged gastric emptying
    - coagulation can become rapidly deranged, especially following amniotic fluid embolus
- foetal concerns
    - foetal distress can occur even if the maternal injury is apparently minor and the mother stable
    - continuous foetal monitoring is required for at least 4-6 hours at >24 weeks gestation, e.g. using cardiotocography (CTG)
        - longer continuous foetal monitoring and obstetric review is required if:
            - uterine contractions
            - non-reassuring foetal heart rate pattern
            - vaginal bleeding
            - significant uterine tenderness or irritability
            - serious maternal injury
            - rupture of the amniotic membranes
    - transplacental haemorrhage
        - all rhesus negative mothers should be given anti-D within 72 hours of injury unless the trauma is trivial or distant from the uterus

    - early obstetric consultation should be obtained where potential uterine or foetal problems are suspected
- trauma considerations
    - foetal injury is more likely in a motor vehicle crash if the lap belt is placed incorrectly across the uterus rather than across the thighs
    - trauma affects up to 7% of pregnancies (only a minority require hospitalisation)
        - specific injuries
            - traumatic brain injury and haemorrhagic shock are the most common mechanisms of death
            - liver and spleen injury and retroperitoneal haemorrhage are all more common
            - bowel injury is less common due to protection from the uterus
        - uterine injury
            - assaults of pregnant women tend to target the uterus
            - uterine injury may co-exist with pelvic fractures
            - uterine injury is rare in blunt trauma (e.g. uterine rupture, placental abruption) but carries the risk of premature labour
                - direct foetal injury occurs in less than 1% of blunt trauma
            - in penetrating trauma, the uterus tends to protect other organs, but foetal mortality is much higher
        - the possibility of domestic violence should always be considered
- resuscitative hysterotomy
    - ideally should be performed within 4 minutes; foetal survival has been reported at >20 minutes
    - applicable for patients known or suspected to be >20 weeks in cardiac arrest
        - earlier than 20 weeks, the foetus is unlikely to survive or have a significant effect on the maternal circulation
    - procedure
        - gloves and apron
        - clean skin and incise from the top of the curve to pubic symphysis vertically
        - make a hole in the peritoneum with the fingers just below the umbilicus and extend with scissors to the symphysis
        - try to avoid the urinary bladder
        - use swabs to help grip and control any bleeding (count if possible)
        - after the incision, identify the uterus
        - vertical or LCSC incision on the lower uterus with the scalpel
            - cutting the baby is not uncommon
            - use plenty of suction
            - complete with scissors
        - deliver the foetus
            - use fundal pressure
            - clamp and cut cord
        - remove the placenta unless it does not come out easily, in which case await for obstetric assistance
        - tranexamic acid

ObC19 OHSS (green-top guideline 5)

- background
    - OHSS is a complication of fertility treatment using pharmacological ovarian stimulation to increase the number of oocytes and therefore embryos available during assisted reproductive technology
        - it may very rarely occur spontaneously in association with pregnancy
    - in a minority of women, the ovarian response exceeds that aimed for
    - there can be significant physical and psychosocial morbidity and even death
        - there is release of cytokines and loss of fluid into the third space and hypovolaemia, with a typical loss of 20% of calculated blood volume in the acute phase
        - there is reduced serum osmolality and sodium
    - in most cases it is self-limiting
    - incidence is unknown as there is no reporting system
- risk factors
    - previous OHSS
    - polycystic ovary syndrome
    - increased antral follicle count
    - high levels of anti-müllerian hormone
    - incidence is higher in cycles where conception occurs, and higher still in multiple pregnancies
- diagnosis
    - symptoms are non-specific and there are no diagnostic tests
        - other serious conditions that present in a similar way must be excluded
    - high level of suspicion required
    - the typical patient presents with abdominal distension and discomfort following the trigger injection used to promote final follicular maturation prior to oocyte retrieval
    - 'early' OHSS presents usually within 7 days of the hCG injection
    - 'late' OHSS presents 10 or more days after the hCG injection
        - late OHSS tends to be more prolonged and severe
- history
    - time of onset of symptoms relative to trigger
    - medication used for trigger (hCG or GnRH)
    - number of follicles on final monitoring scan
    - number of eggs collected
    - whether embryos were replaced and how many
    - any diagnosis of polycystic ovary syndrome
- symptoms
    - abdominal bloating
    - abdominal discomfort/pain, need for analgesia
    - nausea and vomiting
    - breathlessness, inability to lie flat or talk in full sentences
    - reduced urine output
    - leg swelling
    - vulval swelling
    - associated comorbidities such as thrombosis
- differentials

    - pelvic infection
    - pelvic abscess
    - appendicitis
    - ovarian torsion
    - ovarian cyst rupture
    - bowel perforation
    - ectopic pregnancy
- examination
    - general
        - dehydration
        - oedema (pedal, vulval, sacral)
        - heart rate
        - respiratory rate
        - blood pressure
        - body weight
    - abdominal
        - ascites
        - palpable mass
        - peritonism
        - girth
    - respiratory
        - pleural effusion
        - pneumonia
        - pulmonary oedema
- investigations
    - FBC
    - haematocrit (haemoconcentration)
    - CRP (severity)
    - U&Es (hyponatraemia, hyperkalaemia)
    - serum osmolality (hypo-osmolality)
    - liver function tests (elevated enzymes, reduced albumin)
    - coagulation profile (elevated fibrinogen, reduced antithrombin)
    - hCG (to determine outcome of treatment cycle) if appropriate
    - ultrasound (ovarian size, pelvic and abdominal free fluid)
        - consider ovarian doppler if torsion suspected
    - other tests that may be indicated include:
        - ABG
        - d-dimer
        - ECG/echo
        - CXR
        - CTPA or V/Q scan
- assessment of severity
    - mild OHSS
        - abdominal bloating
        - mild abdominal pain
        - ovarian size usually <8cm
    - moderate OHSS
        - moderate abdominal pain

        - nausea +/- vomiting
        - ultrasound evidence of ascites
        - ovarian size usually 8-12cm
    - severe OHSS
        - clinical ascites (+/- hydrothorax)
        - oliguria (<300ml/day or <30ml/hr)
        - haematocrit >0.45
        - hyponatraemia (135 mmol/L)
        - hypo-osmolality (<282 mOsm/kg)
        - hyperkalaemia (>5 mmol/L)
        - hypoproteinaemia (albumin <35 g/L)
        - ovarian size usually >12cm
    - critical OHSS
        - tense ascites/large hydrothorax
        - haematocrit >0.55
        - WCC >25
        - oliguria/anuria
        - thromboembolism
        - acute respiratory distress syndrome
- admission criteria
    - unable to achieve satisfactory pain control
    - unable to maintain adequate fluid intake due to nausea
    - signs of worsening OHSS despite outpatient intervention
    - unable to attend for regular outpatient follow-up
    - critical OHSS
- management
    - analgesia and antiemetics
        - avoid NSAIDs due to renal risk
    - oral fluid replacement guided by thirst
        - severely dehydrated women may require IV fluid replacement and HAS can be considered
    - avoid diuretics
    - paracentesis +/- indwelling drain
        - can be abdominal or vaginal and should be ultrasound guided (to avoid trauma to enlarged, vascular ovaries)
        - indications:
            - severe abdominal distension and abdominal pain secondary to ascites
            - shortness of breath and respiratory compromise secondary to ascites and increased intra-abdominal pressure
            - oliguria despite adequate volume replacement, secondary to increased abdominal pressure causing reduced renal perfusion
- thrombosis
    - women with severe or critical OHSS and those who are admitted should receive LMWH prophylaxis
    - duration should be individualised according to risk factors and outcome of treatment

- thromboembolism should also be suspected in patients with OHSS who present with unusual neurological symptoms, even several weeks after apparent improvement in OHSS

ONCOLOGICAL EMERGENCIES

OncP1 acute presentations of undiagnosed cancer that may present to the ED (including weight loss, dysphagia, pain etc.)

- presentations presenting to the ED include:
  - breast
    - lump in the breast or under the arm
    - nipple changes or discharge
    - skin changes (including erythema, scales, dimpling, puckering)
  - bladder
    - trouble urinating
    - dysuria
    - haematuria
  - spontaneous bleeding or bruising
  - bowel
    - blood in the stools
    - change in bowel habit
  - cough or persisting hoarseness
  - eating
    - pain after eating (persistent heartburn or indigestion)
    - dysphagia
    - abdominal pain
    - nausea and vomiting
    - anorexia
  - fatigue
  - fever or night sweats
  - mouth
    - white or red patch in the mouth or on the tongue
    - bleeding, pain or numbness of the lip or mouth
  - neurological
    - headaches
    - seizures
    - vision changes
    - hearing changes
    - facial droop
  - dermatology
    - flesh coloured lump that bleeds or scales
    - new mole or change in existing mole
    - non-healing sore
    - jaundice
  - swelling or lumps
  - weight gain or weight loss for no reason

OncC1 complications related to local tumour progression e.g. acute cord compression, upper airway obstruction, pericardial and pleural effusions, SVC compression syndrome, raised intracranial pressure

- acute metastatic cord compression
    - background
        - caused by pathological vertebral body collapse or direct tumour growth causing compression of the spinal cord
        - majority is either thoracic (60-70%) or lumbar (20-30%)
        - a small proportion (5%) present in the cervical spine
    - presentation
        - patient may or may not have a cancer diagnosis/primary disease
        - referred back pain that is multi segmental or band like
        - escalating pain, poorly responsive to treatment, including medication
        - different character or site to previous symptoms
        - funny feeling, odd sensations or heavy legs (multi-segmental), pins and needles
        - lying flat increases back pain
        - pain, worse on coughing or sneezing
        - agonising pain causing anguish and despair
        - gait disturbance, unsteadiness, especially on stairs (not just a limp)
        - sleep grossly disturbed due to pain being worse at night
        - established motor/sensory/bladder/bowel disturbances or incontinence are late signs
            - if the lesion is above T12, the patient will have an upper motor neurone (spastic) bladder
                - incontinence
                - no voluntary control of bladder emptying
            - if the lesion is below T12, the patient will have a lower motor neurone (flaccid) bladder
                - urinary retention
                - dribbling incontinence when bladder is overfilled
                - large post voiding residual volumes
    - initial ED management
        - investigation
            - urgent MRI spine
            - bloods
                - FBC (anaemia)
                - U&E
                - LFT (derangement suggesting liver metastasis)
                - CRP (spinal infection)
                - calcium
        - treatment
            - 16mg dexamethasone as soon as possible after assessment
            - blood glucose monitoring and gastric protection
            - observe for signs of autonomic dysreflexia
                - stimulus below the level of the spinal lesion (e.g. full bladder) causes uncoordinated autonomic response

                - assess spine and neurology regularly
                - nurse flat, log roll until bony and neurological stability ensured
                - consider catheter as an unstable spine may not allow for a bed pan
                - bowel management
                - pain management
                - thromboprophylaxis
                - nil by mouth if surgery is a possibility
                - further treatment depending on MRI result
        - grading
            - grade 1
                - mild paraesthesia
                - subjective weakness
                - no objective findings
            - grade 2
                - mild or moderate sensory loss
                - moderate paraesthesia
                - mild weakness with no loss of function
            - grade 3
                - severe sensory loss, paraesthesia or weakness that interferes with function
            - grade 4
                - paralysis
- management
    - grade 1
        - rule out spinal cord compression – full neurological examination
        - discuss with acute oncology team
        - MRI whole spine to be performed within one week of clinical suspicion
        - patient should not be discharged until you are sure they do not have MSCC
        - if they are discharged they should have review within 24 hours and 24h contact if symptoms worsen/persist
    - grade 2, 3, 4
        - rule out spinal cord compression/cauda equina: urgent MRI whole spine
        - treat as unstable spine until MRI results reported or oncology/neurosurgical assessment
        - admit for monitoring and ongoing assessment
        - commence:
            - dexamethasone 16mg stat dose then 16mg OD
            - analgesia
            - thromboprophylaxis
        - if no previous known malignancy and lymphoma suspected, steroids should be avoided before biopsy if possible (may cause rapid resolution of tumour and make histological diagnosis difficult)
        - MSCC co-ordinator or on-call oncologist should be contacted to assess and plan treatment (radiotherapy or surgery if required)
        - all patients should start definitive treatment within 24 hours of MRI confirmation of diagnosis
- upper airway obstruction

- background (central airway obstruction)
  - refers to a variety of obstructive processes that impede airflow within the central airways, trachea and mainstem bronchi
  - may present in a variety of ways
  - frequently misdiagnosed – high level of suspicion required
  - may be malignant or non-malignant
- history and examination
  - presence of risk factors
  - shortness of breath
    - late sign
    - constant
    - occasionally positional (worse recumbent)
    - not responsive to bronchodilators
  - cough
    - normally chronic, persistent and dry
  - haemoptysis
    - common, especially in tracheal lesions and may be massive
  - wheeze
    - may be inspiratory or expiratory
    - location of wheeze does not always correspond to location of obstruction
    - unilateral wheeze suggests obstruction distal to the carina
    - wheeze may be positional and unresponsive to bronchodilators
  - stridor
    - develops when the airway diameter is <5mm
    - represents severe subglottic or tracheal stenosis
    - inspiratory stridor suggests extrathoracic airway obstruction at or above the vocal cords and is best heard over the neck
    - expiratory stridor may be due to an intrathoracic obstruction
    - biphasic stridor is present in subglottic or tracheal stenosis
    - manoeuvres that increase airflow, such as hyperventilation, may accentuate the stridor, and neck flexion may change the intensity
  - other diagnostic factors (uncommon)
    - hoarseness
    - orthopnoea
      - due to structural compression by large intrathoracic tumours
    - dysphagia
    - chest pain
    - anxiety
      - may herald an impending respiratory arrest
    - tachypnoea
      - may herald an impending respiratory arrest
    - tachycardia
      - may herald an impending respiratory arrest
    - accessory muscle use
      - may herald an impending respiratory arrest

        - cyanosis
            - may herald an impending respiratory arrest
        - crackles
- risk factors
    - strong
        - lung cancer
            - most common cause is direct extension from an adjacent tumour
        - primary airway malignancy
        - smoking
        - artificial airways
            - risk of granulation tissue, tracheomalacia and subglottic or tracheal stenosis
        - tracheobronchial stents
        - transtracheal oxygen catheters
        - lung transplantation
        - neurocognitive and neuromuscular disorders
            - risk of foreign body aspiration
        - relapsing polychondritis
        - granulomatosis with polyangiitis (Wegener's)
        - tracheobronchomalacia
    - weak
        - endobronchial infections
            - e.g. TB, histoplasmosis
        - extrathoracic and distant malignancies
- investigations
    - CXR
        - should be obtained for every patient
        - infrequently diagnostic
        - air trapping is the hallmark of airway obstruction on CXR
            - failure of the lung to decrease in volume and increase in opacity on exhalation compared with inhalation
            - some mediastinal shift to the side that is not trapping
        - there may be post-obstructive pneumonia, atelectasis, lobar or total collapse
    - bronchoscopy (flexible or rigid)
        - most specific and sensitive test
        - provides information on the location and morphology of the lesion, the amount of intraluminal disease and extraluminal compression, and the diameter and length of the lesion
        - tissue can be obtained for biopsy
        - potentially dangerous in severe obstruction, as the bronchoscope further narrows the lumen
        - swelling or bleeding may occur post procedure
        - sedation may create an unstable airway
    - CT chest

- allows determination of the type of obstructive lesion and the patency of the airway distal to the obstruction, the length and diameter of the lesion and its relationship to nearby structure, including vessels
- also allows 3D reconstruction

- MRI chest
  - can assess the larynx, proximal trachea and mediastinal and hilar masses and to differentiate between vascular and soft tissue masses
- flow-volume loops
  - allows identification and categorisation of upper airway obstruction
- spirometry
  - should not be undertaken in patients with respiratory distress or advanced CAO due to the possibility of inducing respiratory failure
- endobronchial ultrasound
  - emerging as an investigation

- management
  - acute
    - establishment of secure airway
      - options include:
        - ET intubation
        - rigid bronchoscopy
        - fibre-optic assisted intubation
        - LMA
        - cricothyroidotomy
        - tracheotomy
      - adjuncts include:
        - heliox (60-80% helium and 20-40% oxygen)
          - reduces turbulent flow of gases in the large airways and therefore reduces work of breathing
          - may be used a bridge to allow a more stable intubation
          - cannot deliver FiO2 >40%
        - diagnostic flexible bronchoscopy once stabilised
        - endoscopic airway intervention
          - thermal
            - laser therapy
            - electrosurgery
            - argon plasma coagulation
            - cryotherapy
          - non-thermal
            - photodynamic therapy
            - airway dilation with rigid bronchoscope or balloon bronchoplasty
            - airway stenting
            - endobronchial microdebrider

- radiation
    - brachytherapy
- external beam radiation
- surgery

- subacute presentation
    - supplemental oxygen
    - bronchodilators
    - continuous positive pressure ventilation
    - diagnostic flexible bronchoscopy
    - endoscopic airway intervention
    - external beam radiation
    - surgery

- pericardial and pleural effusions
    - malignant pericardial effusion
        - background
            - an accumulation of fluid within the pericardial sac leading to an effusion can be a presenting symptom in acute oncology patients
            - two thirds of cancer patients have subclinical pericardial effusions with no overt cardiovascular signs or symptoms
            - 50% of cases initially present with symptoms of cardiac tamponade
            - symptoms are often attributed to underlying cancers and are often a pre-terminal event, however prompt diagnosis and management can achieve significant palliation
        - causes
            - most malignant pericardial effusions result from direct malignant involvement with the pericardium
            - other, rarer causes include:
                - radiation induced pericarditis
                - chemotherapy induced pericarditis (e.g. doxorubicin or cyclophosphamide)
        - clinical findings
            - dyspnoea (majority)
            - fatigue
            - asthenia (abnormal weakness/fatigue)
            - cough
            - chest pain
            - orthopnoea
        - examination findings
            - elevated JVP
            - tachycardia
            - hypotension
            - pulsus paradoxus (abnormally large decrease in pulse and systolic blood pressure >20mmHg with inspiration)
            - Kussmaul's sign -increased distension of jugular veins with inspiration
        - diagnosis
            - CXR

        - widened cardiac shadow
    - echo
        - shows size of effusion and haemodynamic compromise
    - ECG
        - small ECG complexes
- differential
    - chest infection
    - PE
    - disease progression
    - ascending aortic aneurysm due to indwelling intravascular catheter
- grades
    - grade 1
        - small effusion with no haemodynamic compromise
    - grade 2
        - moderate effusion with no or minimal haemodynamic consequence and good left ventricular function
    - grade 3
        - effusion with haemodynamic consequence
    - grade 4
        - cardiac tamponade – life-threatening consequences, urgent intervention required
- management
    - grade 2
        - enquire about signs of sepsis/productive cough and escalate to grade 3 as required
        - discuss with acute oncology team prior to discharge for ambulatory management
        - early referral to cardiology for management advice
        - review after 24 hours and provide contact if worsening
    - grade 3 and 4
        - admit for ongoing assessment, monitoring and symptom management
        - withhold anticoagulation
        - consider immediate therapeutic drainage if cardio-vascular compromise
        - urgent referral to cardiology or cardiothoracic on call team
        - inform acute oncology team
        - all treatment options should be balanced against the patient's symptoms, overall performance status, level of disease and predicted benefits

- malignant pleural effusion pathway
    - proven malignant pleural effusion
        - if not symptomatic, observe unless drain required for other reasons
        - if symptomatic:
            - if long life expectancy and limited systemic disease, consider referral to thoracic surgeons for thoracoscopic drainage/pleurodesis/PleurX or other indwelling catheter

        - if not, and systemic therapy unlikely to lead to rapid resolution – insertion of intercostal tube and drainage
            - if systemic therapy likely to lead to rapid resolution, requires urgent oncology review for chemotherapy
    - some places have an ambulatory pathway available for management of stable patients requiring drainage of pleural effusion
- SVC compression syndrome
    - background
        - obstructive emergency that may occur as the result of progression of malignancy of may be the diagnostic symptom
        - caused by external pressure, thrombus or direct tumour invasion causing obstruction of the superior vena cava and occurs in 3-8% of patients with cancer
        - impairs venous return to the right atrium
    - anatomy
        - the SVC is the principle venous drainage to head, neck and upper extremities
        - the main collateral is the azygos vein, which joins the SVC posteriorly over the right main bronchus and drains the posterior thorax
        - if obstruction occurs distal to the azygos insertion, compensation occurs
        - if obstruction occurs proximal to the azygos, flow must bypass the SVC and return via the internal mammary, superficial thoracoabdominal, vertebral venous system to the IVC, resulting in very high pressures
    - differential
        - chest infection
        - PE
        - disease progression
        - ascending aortic aneurysm due to indwelling intravascular catheter
    - signs and symptoms
        - dyspnoea
        - stridor – due to laryngeal oedema
        - non-pulsatile JVP
        - headaches
        - dilated anterior chest wall veins
        - confusion
        - chest pain
        - swelling of face and neck
        - coma
    - investigations
        - CTPA to define tumour extent, site of occlusion or stenosis and extent of any thrombus
            - SVCO can be an incidental finding on CT
    - grading
        - grade 1
            - oedema in head or neck
            - vascular distension
            - cyanosis
            - plethora

        - grade 2
            - oedema in head or neck with functional impairment (mild dysphagia, cough, visual disturbance)
        - grade 3
            - mild or moderate cerebral oedema (headache, dizziness)
            - mild or moderate laryngeal oedema or diminished cardiac reserve (syncope after bending)
        - grade 4
            - significant cerebral oedema (confusion)
            - significant laryngeal oedema (stridor)
            - significant haemodynamic compromise
    - management
        - grade 2
            - enquire about signs of sepsis/productive cough and escalate to grade 3 as appropriate
            - consider steroid therapy to manage symptoms of oedema and prevent deterioration
            - enquire about history of underlying chest complaints and advise on management
            - arrange acute oncology and/or respiratory review
        - grade 3 or 4
            - admit for further assessment and management
            - monitor for evidence of:
                - desaturation
                - infection
                - SACT/chemotherapy toxicities
                - haemodynamic compromise
            - address life-threatening symptoms such as stridor
            - initial treatment is aimed at symptom management
            - commence high dose steroids and PPI if not contraindicated
            - urgent acute oncology and/or respiratory team guidance
        - further management
            - may include (depending on performance status and contraindications):
                - stent insertion
                - chemotherapy
                - radiotherapy
                - anticoagulant if thrombus present and no contraindications
- raised intracranial pressure
    - history
        - cancer diagnosis/primary disease/known metastatic disease
        - currently receiving or recently completed SACT treatment
        - currently receiving or recently completed radiotherapy
        - whether the presenting symptoms are new
        - any co-existing conditions such as epilepsy, hypertension, medication that may account for symptoms
    - clinical/neurological assessment

        - new onset of seizures
        - headache
        - visual disturbance
        - nausea and/or vomiting
        - cognitive dysfunction
        - confusion
        - disorientation and/or memory loss
        - motor dysfunction
        - symptoms of stroke
    - investigation
        - FBC, U&E
        - CT head +/- MRI
    - management
        - dexamethasone
            - if serious neurological impairment/requiring admission – 16mg PO OD(IV if needed) with PPI cover
            - less serious neurological impairment likely to require home care/nursing assistance - 8-16mg PO OD
        - antiepileptic medication if having convulsions
        - admission for monitoring, ongoing assessment and management
        - early involvement from acute oncology team
        - early critical care management/advice if there is deterioration
        - patients with no known malignancy require neurosurgical referral
        - patients with known primary disease presenting with metastatic disease require referral to the brain and CNS MDT

OncC2 complications relating to cancer treatment including neutropenic sepsis, anaemia and thrombocytopaenia, and immunotherapy

- neutropenic sepsis
    - background
        - febrile neutropenia is defined as a single temperature ≥38.5 or sustained temperature of ≥ 38 for more than an hour in a patient with neutrophils <0.5 x109/L or <1 with predicted nadir of <0.5 over the subsequent 48 hours
        - patients with potential neutropenia (e.g. received or receiving SACT, history of myelosuppression or bone marrow failure) and clinical suspicion of infection should be treated immediately
        - neutropenic patients with sepsis may not have a fever
        - rapid empirical initiation of broad spectrum antibiotics and source control is essential
        - non-infectious causes of fever in patients with haematological malignancy are common but cannot be distinguished in the acute setting
    - infective organisms
        - main organisms are:
            - Gram negative bacilli
            - coagulase-negative Staphylococci
            - Staphylococcus aureus
            - Streptococcus viridans

        - others
            - resistant organisms in some patient groups/institutions
            - fungal infection
    - antibiotic choice
        - needs to cover likely causative organisms
        - choice depends on local susceptibility patterns and patient risk of infection with a multi-drug resistant organism
        - first line is Tazocin 4.5g TDS (QDS if septic shock/critically ill)
        - other possible antibiotics
            - gentamycin IV 4-7mg/kg IBW if risk of resistance or patient critically ill
                - avoid aminoglycosides in patients who have received platinum based chemotherapy in the previous week
            - vancomycin
                - for suspected MRSA
                - in severe sepsis/septic shock
                - known to be colonised with MRSA
                - clinical evidence of catheter-related infection in a unit with high incidence of MRSA
                - persistent fever at 48 hours
            - meropenem
                - for suspected ESBL
            - antifungal
                - suspected fungal infection
                - on expert advice when fever persists in high risk patients after 96 hours of antibiotic therapy
            - co-trimoxazole
                - suspected PCP
            - acyclovir
                - suspected HSV/CMV
    - risk factors for neutropenic sepsis
        - post-chemotherapy
        - post-transplantation
        - chronic granulomatous disease
        - clozapine induced agranulocytosis
    - assessment
        - upper respiratory tract
            - otitis media
            - sinusitis
        - oropharynx
            - dental abscess
            - mucositis
        - lower respiratory tract
            - signs of pneumonia
        - abdomen
            - signs of Clostridium difficile colitis (generalised abdominal tenderness)

        - typhlitis (tenderness over caecum)
    - skin
        - cellulitis
        - vesicular lesions
    - perineum and perianal area
        - anal fissure
        - cellulitis
        - abscess
        - do not perform a rectal examination
    - CVAD (central venous access device)
        - signs of tunnel/exit site infection
    - signs of anaemia and/or thrombocytopenia
- investigations
    - blood cultures
        - plus cultures from any existing intravascular devices
    - FBC, U&E, LFT, CRP, lactate
    - if clinically indicated:
        - CXR
        - nasal/throat swab for respiratory virus PCR
        - sputum microscopy and culture
        - stool culture and viral studies if diarrhoea; C difficile toxin assay if recent antibiotic treatment
        - bacterial swab of skin, vascular access site or mouth lesions
        - viral swab of vesicular lesions and mouth ulcers
        - CT head and LP if neurological symptoms
        - CT pan scan if occult infection source
        - echo (endocarditis)
- management
    - resuscitation
        - address septic shock
            - IV fluid resuscitation
            - vasopressor support
            - invasive monitoring
    - IV antibiotics
        - within 1 hour, preferably within 30 minutes if septic
    - early source identification and control
    - environmental precautions
        - protective isolation
        - hand hygiene
        - full barrier precautions
    - G-CSF
        - increases neutrophil count
        - reduces rate of serious infections
        - should be stopped once neutrophils >1.0
        - adverse effects: rash, injection site pain, bone pain, influenza-like symptoms, splenic rupture (rare)
- prognosis

        - general
            - proportional to the number of organ failures involved
        - factors improving ICU survival
            - better patient selection
            - early admission before the onset of multi-organ failure
            - overall improved prognosis in haematological and solid malignancy
            - use of NIV
            - use of diagnostic bronchoscopy
            - improved survival rates in septic shock
- anaemia
    - background
        - anaemia in cancer patients is a common cause of debilitating fatigue and impaired quality of life
        - causes include:
            - infiltration of bone marrow by malignant cells
            - suppression of red cell production by inflammatory cytokines
            - cytotoxic chemotherapy
            - nutritional deficiencies
            - renal damage
            - bleeding
    - management
        - red cell transfusions or erythropoietin-stimulating agents (ESAs)
        - decision to treat should be based on symptoms such as fatigue, breathlessness and impaired quality of life rather than a specific Hb concentration
        - relief of symptoms is the target of therapy
        - radiotherapy may be less effective in the presence of anaemia (hypoxic cancer cells are less sensitive to radiation) but the benefit of red cell transfusion to reduce mild anaemia in this setting is controversial
        - treatment with ESAs can modestly reduce blood transfusion exposure and improve quality of life in selected patients, especially in those treated with nephrotoxic platinum compounds
            - there is evidence that ESA can promote the growth of a range of non-haematological tumours
            - ESAs may increase the already raised VTE risk
        - maximum improvement in fatigue and quality of life occurs between 110 and 120g/L
- thrombocytopenia and bleeding
    - background
        - bleeding can occur secondary to disease, injury or as a side effect of treatment
        - thrombocytopaenia is a reduction in the number of platelets
            - if <50, bleeding or bruising may occur with minor trauma
        - in a non-septic patient, a platelet count of 10 or above may be adequate in the absence of additional risk factors for bleeding
    - pathophysiology

        - thrombopoietin produced by the liver and kidneys stimulates thrombopoiesis
        - platelets are derived from megakaryocytes
        - average platelet lifespan is 9 days; destruction occurs by phagocytosis in the spleen and liver
        - decreased production generally leads to a gradual fall in platelets over a week
        - immune destruction typically leads to an abrupt fall
        - the spleen normally sequesters 1/3 of the body's platelets, which increases to 90% in massive splenomegaly
        - spleen contraction stimulated by the sympathetic nervous system can release additional platelets
    - general causes of thrombocytopenia
        - decreased production due to bone marrow failure (myelosuppression or infiltration)
            - drugs (temporal association with starting a drug, typically improves after a few days)
            - alcohol
            - infection
            - nutritional (e.g. B12, folate, copper)
            - infiltration
            - haematological disorders (e.g. leukaemia)
            - liver cirrhosis (decreased thrombopoietin)
        - increased destruction
            - sepsis
            - immune (ITP)
            - intravascular device
            - drug induced (antibiotics, thiazides, H2 antagonists)
            - antiphospholipid syndrome
            - TTP
            - HUS
            - DIC
            - HELLP
            - haemolysis
            - massive haemorrhage
        - increased aggregation
            - early sign of multi-organ dysfunction
            - sepsis
            - massive PE
            - thrombotic storm (widespread thrombosis in multiple organs)
        - dilution
            - post resuscitation
            - massive transfusion
        - sequestration
            - splenomegaly
        - spurious
        - often multifactorial

- history
  - cancer diagnosis/primary disease
  - taking anticancer treatment at the moment or recently, if so what and when it stopped
  - any active bleeding
    - site
    - injury related or spontaneous
  - amount of blood lost
  - onset and duration
  - any previous similar bleeding or bruising
  - allergies and current medications
  - any relieving factors including whether it is stopped by direct pressure or other measures
- associated symptoms
  - light-headedness
  - pallor
  - clamminess
  - thirst
  - rash (petechial, purpuric, punctuate)
- investigations
  - FBC
  - U&Es
  - G&S/crossmatch
  - coagulation
  - fibrinogen if considering DIC
- grading
  - grade 1
    - bleeding: mild, self-limiting, controlled by conservative measures
    - ecchymosis
    - occult blood in secretions
    - bruising: petechiae or bruising in a localised or dependant area, with or without trauma
  - grade 2
    - bleeding: blood loss of 1-2 units
    - bruising: moderate petechiae, purpura and/or generalised bruising, with or without trauma
  - grade 3
    - bleeding: blood loss of 3-4 units
    - bruising: generalised petechiae, purpura and/or bruising
    - new bruises without significant trauma
  - grade 4
    - massive bleeding – blood loss >4 units
    - life-threatening haemorrhage
- management
  - grade 1
    - review blood results
    - manage neutropenia as needed

                - discuss abnormalities with on call haematologist or oncologist
            - grade 2, 3, 4
                - resuscitate as required
                - stop any contributing medication if safe to do so
                - reversal of anticoagulants if needed
                - discuss with haematologist or oncologist
                - admit and consider critical care admission
            - platelets may be required if invasive procedures are needed or there is intracranial haemorrhage
- immunotherapy
    - immune-related adverse events
        - adrenal crisis
            - associated with immune checkpoint inhibitors
            - signs and symptoms
                - hypovolaemic shock
                - hypotension (systolic <90mmHg)
                - postural hypotension
                - dizziness/collapse
                - nausea/vomiting
                - abdominal pain/tenderness/guarding
                - fever
                - confusion/delirium/coma
                - hyponatraemia/hyperkalaemia
                - hypoglycaemia
                - pre-renal failure
            - endocrine function panel required:
                - TSH
                - free T4
                - ACTH
                - cortisol
                - prolactin
                - blood glucose
                - LH
                - FSH
                - testosterone/oestrogen
            - immediate administration of 100mg hydrocortisone IV or IM
                - followed by continuous infusion of 200mg per 24h (or 50mg IV/IM every 6 hours)
            - rehydration with 1 litre saline in the first hour, then as required (usually 6 litres/24 hours)
            - urgent endocrinology referral
            - withholding of next cycle of ICPi
        - hypophysitis
            - presentation
                - headache
                - fatigue
                - visual loss

        - non-specific symptoms:
            - nausea
            - diarrhoea
            - malaise
            - anorexia
        - low threshold for clinical suspicion required
    - investigation
        - endocrine function panel
        - FBC, U&E, LFTs
        - MRI of the pituitary
        - formal visual field assessment
    - management
        - depends on severity
            - cortisol and/or thyroxine replacement according to endocrinology guidance
            - start steroids after sending endocrine function panel
- thyroid dysfunction
    - background
        - typical pattern of thyroid specific biochemical disturbance presenting with asymptomatic hyperthyroidism before return to normal levels for a brief period
            - nearly always followed by hypothyroidism, sometimes profound, frequently persistent, requiring long-term thyroid replacement
    - hyperthyroidism
        - TSH <0.4 and free T4 >upper limit of normal
        - check TSH receptor antibodies
        - symptoms
            - fatigue or muscle weakness
            - hand tremor
            - mood swings
            - nervousness or anxiety
            - rapid heartbeat
            - heart palpitations or irregular heartbeat
            - skin dryness
            - trouble sleeping
            - weight loss
            - increased frequency of bowel movements
            - menstrual disturbance
        - management if persisting includes
            - propranolol or atenolol
            - prednisolone for painful thyroiditis
            - consider carbimazole on endocrinology advice
    - hypothyroidism
        - TSH >10 and free T4 <lower limit of normal
        - symptoms
            - fatigue
            - weakness

                - sensitivity to cold
                - weight gain or difficulty losing weight
                - coarse dry hair and dry skin
                - hair loss
                - muscle cramps and aches
                - constipation
                - depression
                - irritability
                - memory loss
                - abnormal menstrual cycles
                - decreased libido
                - in severe cases:
                    - slowed speech
                    - jaundice
                    - increased tongue size
        - management
            - levothyroxine 75 microgram (or 25 microgram for high risk patients – profound hypothyroidism, cardiac conditions, elderly)
- gastrointestinal
    - background
        - among the most common immune related adverse events
        - can become life-threatening if unrecognised and untreated
        - can be delayed effects up to 12 months after completion of treatment
    - grade 1 – mild
        - <4 stools/day over baseline or mild increase in ostomy output
        - no history of abdominal pain, mucous/blood in stool
        - investigations
            - baseline bloods
            - stool microscopy and culture
            - C diff toxin
            - faecal calprotectin if symptoms persist
        - management
            - loperamide
            - encourage fluids
            - avoid high fibre and lactose
    - grade 2 – moderate
        - any of the following:
            - 4-6 stools/day over baseline or moderate increase in ostomy output
            - moderate abdominal pain/cramps/discomfort
            - mucous in stool regardless of number of stools
            - nocturnal stools
            - persistent grade 1 symptoms

        - investigations as above plus PCR screen and AXR +/- CT abdo/pelvis
        - management
            - prednisolone 0.5-1mg/kg/day and PPI cover
            - stop loperamide and codeine (increased risk of colonic dilatation and perforation)
            - fluid balance and replacement as required (including dioralyte)
    - grade 3 and 4 – high
        - any of:
            - ≥6 stools/day over baseline or significant increase in ostomy output
            - severe abdominal pain
            - fever
            - dehydration
            - blood in stool
            - incontinence
            - limitation of ADLs
            - episodes within 1 hour of eating
        - investigations as above plus flexible sigmoidoscopy
        - management
            - gastroenterology review
            - IV hydration and fluid balance
            - IV methylprednisolone 1-2mg/kg/day and PPI cover
            - stop loperamide and codeine
- hepatic
    - background
        - hepatic transaminases (ALT/AST) and bilirubin should be checked before each dose of immunotherapy
    - investigations
        - AST/ALT
        - GGT
        - CK
        - amylase
        - INR
        - hepatitis PCR
        - ferritin
        - EBV/CMV
        - HSV/parvovirus
        - lactate and ABG/VBG for assessment of acidosis
        - autoimmune screen
        - hepatitis ABCE screen
        - direct bilirubin if bilirubin markedly elevated
        - FBC
        - US scan in the first 12-24 hours to assess spleen size and doppler to assess flow in hepatic veins and arteries
    - management
        - monitoring

        - steroids
        - medication review – stop regular paracetamol and NSAIDs and all non-essential medication
        - CT +/- biopsy may be required in severe cases
- neurological
    - background
        - neurological immune-related adverse events can manifest as central abnormalities (e.g. aseptic meningitis, encephalitis) or peripheral sensory/motor neuropathies (e.g. Guillain-Barré)
        - early recognition and management is important and can be difficult as neurological symptoms can be common in people with cancer
    - grade 1
        - asymptomatic
        - loss of deep tendon reflexes or paraesthesia
        - not interfering with function or ADLs
        - not concerning to patient
        - investigations:
            - full neurological examination
            - review of alcohol and medication history
            - FBC, U&E, ALT, cortisol, TFTs, glucose, B12, folate, HIV, TSH
            - consider autoimmune and vasculitis screen
            - consider MRI/MRA brain/spine +/- CSF
    - grade 2
        - sensory alteration or paraesthesia
        - some interference with function
        - cranial nerve problem
        - symptoms concerning to patient
        - investigations
            - as above and consider nerve conduction studies for lower motor neurone and/or sensory changes
            - consider pulmonary function and diaphragmatic function tests
        - management
            - daily monitoring
            - consider IV methylprednisolone or oral prednisolone
            - consider pregabalin or duloxetine for neuropathic pain
            - consider orthotic devices
    - grade 3 and 4 - severe or life-threatening
        - severe or disabling symptoms
        - limiting self care and ADLs
        - life threatening (e.g. respiratory) problems
        - investigations
            - as above

                - plus consider muscle biopsy
        - management
            - urgent neurology review
            - permanently discontinue ICPi
            - IV hydration
            - IV methylprednisolone 1-2mg/kg/day and PPI cover
            - treatment for specific disorders
- pneumonitis
    - background
        - can occur after a single dose up to as many as 48 treatments
        - incidence may be greater with immunotherapy combination therapies
    - grade 1 (mild)
        - clinically asymptomatic
        - radiographic changes only
            - e.g. focal ground glass opacities, patchy infiltration
        - investigation
            - sputum for MC&S and PCP PCR
            - baseline bloods
            - CXR
            - CT
        - management
            - monitor and re-image if worsening
    - grade 2
        - mild to moderate
        - new onset of symptoms limiting ADLs
        - e.g. dyspnoea, cough, fever, chest pain
        - investigations
            - as above plus bronchoscopy +/- lavage
        - management
            - if no evidence of infection, prednisolone 60mg/day and PPI cover
            - antibiotics if evidence of infection
    - grade 3 and 4
        - severe or life-threatening
        - severe new onset of symptoms limiting self care
        - or hypoxia
        - or ARDS
        - investigations
            - as above +/- pulmonary function tests
            - exclude atypical infections
                - galactomannan and beta-D-glucan
                - urine legionella and pneumococcal antigen
                - mycoplasma serology
                - pneumocystis jiroveci screening
        - management
            - IV methylprednisolone 2mg/kg/day and PPI

                - can be increased to 4mg/kg/day
            - consider empirical antibiotics and antifungals
- renal toxicity
    - background
        - tubulointerstitial nephritis and allergic nephritis have infrequently been observed
    - grade 1
        - creatinine <1-1.5 x increase from baseline (stage 1 AKI)
    - management
        - weekly creatinine monitoring
        - good hydration
        - stop nephrotoxic drugs
    - grade 2
        - creatinine >1.5 and ≤3 x increase from baseline (stage 2 AKI)
        - investigations
            - FBC, U&E, LFT, cortisol, TFTs, glucose
            - urinalysis
                - protein positive – check protein creatinine ratio
                - blood positive – send to microbiology for casts
                - blood and protein – send for both
        - management
            - if clear urinalysis, good hydration, hold nephrotoxics, repeat bloods in 24-48 hours
            - if positive urinalysis, 1mg/kg/day oral prednisolone and PPI
            - referral to renal team
    - grade 3 and 4
        - creatinine >3x upper limit of normal (stage 3 AKI)
        - investigations
            - as above plus daily weight, fluid balance, exclude other causes
        - management
            - admit
            - IV methylprednisolone 1-2mg/kg/day and PPI
            - IV hydration as indicated
- skin toxicity
    - background
        - common, but typically mild to moderate severity
    - grade 1 and 2
        - skin rash affecting <10% body surface area up to 30%, with or without symptoms
        - investigations
            - FBC, U&E, LFT, cortisol, TFTs, glucose
            - photograph rash and measure lesions
        - management

        - antihistamines – topical or oral
        - topical steroids
        - topical emollient
    - grade 3
        - skin rash affecting >30% of body surface area or grade 2 with substantial symptoms
        - investigations
            - as above
        - management
            - mild to moderate: prednisolone 5mg/kg/day for 3 days then taper over 1-2 weeks, PPI
            - severe: IV methylprednisolone 0.5-1mg/kg/day, converted to oral on response, tapered over 2-4 weeks
            - antihistamines
            - emollient with paraffin content
        - grade 4
            - skin sloughing >30% body surface area with associated symptoms:
                - generalised
                - exfoliative
                - ulcerative
                - bullous
                - purpura
            - investigations
                - as above plus punch biopsy and fluid balance
            - management
                - urgent dermatology review
                - IV hydration in extensive bullous/desquamating rashes
                - IV methylprednisolone 1-2mg/kg/day plus PPI
                - antihistamines
                - emollients
                - analgesia

OncC3 biochemical complications of malignancy – hypercalcaemia, SIADH, adrenocortical insufficiency

- hypercalcaemia
    - background
        - a disorder characterised by laboratory test results that indicate an elevation in the concentration of calcium (corrected for albumin) in blood
        - corrected calcium >3.4 mmol/L requires urgent treatment
    - history
        - whether there is a cancer diagnosis/primary disease
        - whether they are taking anticancer treatment at the moment/recently

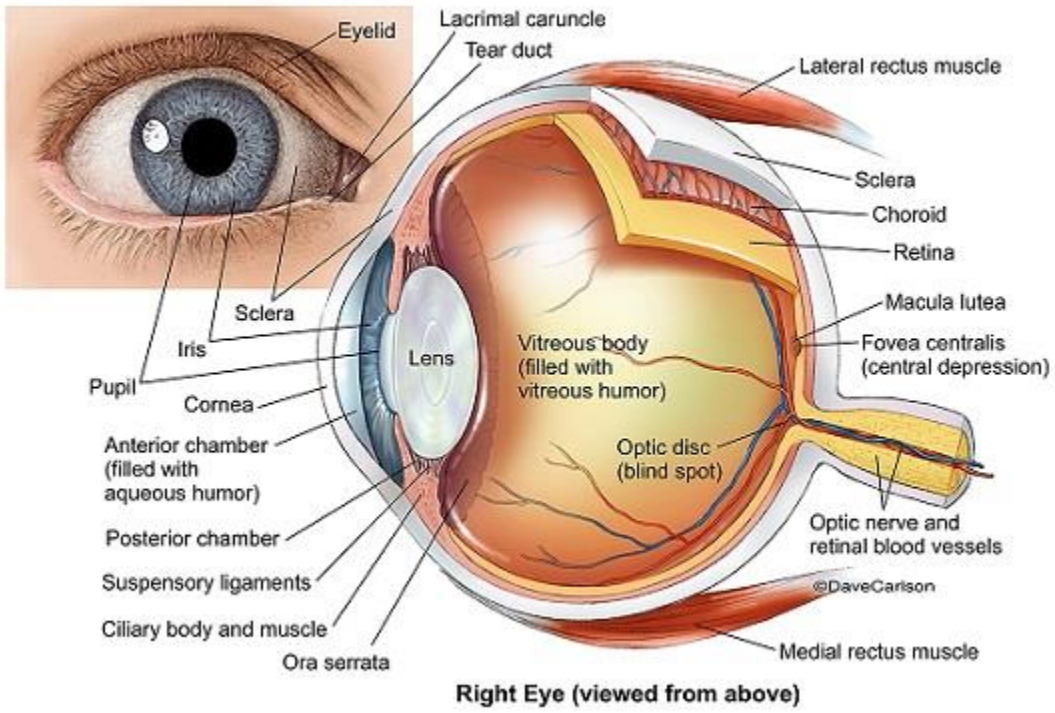
        - whether they have previously had hypercalcaemia
        - whether they are taking any other medication
    - investigations
        - ECG
            - shortened QT interval or other conduction abnormalities
        - bloods
            - calcium adjusted for albumin, phosphate, PTH, vitamin D, U&Es
        - signs and symptoms
            - polyuria and thirst
            - anorexia
            - nausea/vomiting
            - constipation
            - abdominal pain
            - fatigue/lethargy
            - mood disturbance
            - cognitive dysfunction
            - confusion
            - seizures
            - renal impairment
            - pancreatitis
            - peptic ulceration
            - muscle weakness
            - band keratopathy (corneal calcium deposits)
            - hypertension
            - cardiomyopathy
            - shortened QT interval
            - dysrhythmias
            - coma
        - grade 1
            - corrected calcium >upper limit of normal – 2.9 mmol/L
        - grade 2
            - >2.9 – 3.0 mmol/L
            - often asymptomatic and does not usually require correction
        - grade 3
            - >3.0 – 3.4 mmol/L
            - may be well tolerated if risen slowly but may be symptomatic and prompt treatment is usually indicated
        - grade 4
            - >3.4 mmol/L
            - requires urgent correction due to risk of dysrhythmia and coma
        - management
            - 2-4 litres of 0.9% sodium chloride IV
            - followed by 4mg zoledronic acid IV in 100ml 0.9% sodium chloride
                - do not give bisphosphonate if creatinine clearance <30ml/min (GFR <10) – seek advice
                - zoledronic acid needs to be reduced in renal impairment

        - repeat dose of bisphosphonates should not be given for at least 4 days after previous dose as maximum effect not seen yet and risk of hypocalcaemia
    - endocrinology advice
    - review medications that may affect renal blood flow
    - recheck U&Es and calcium after 4-7 days or sooner if needed to monitor fluid replacement
- SIADH
    - background
        - hyponatraemia due to an increase in concentration of ADH inappropriate to the current osmotic or volume status
    - diagnostic criteria
        - hypotonic hyponatraemia
        - urine osmolality > plasma osmolality – concentrated urine despite hypotonic blood
        - urinary sodium >20mmol/L
        - normal renal, hepatic, cardiac, pituitary, adrenal and thyroid function
        - euvolaemia (absence of hypotension, hypovolaemia, oedema)
        - correction by water restriction
    - causes (MAD CHOP)
        - major surgery
            - abdominal
            - thoracic
            - transsphenoidal pituitary (6-7 days post-op)
        - ADH production by tumours (ectopic)
            - small cell bronchogenic carcinoma
            - adenocarcinoma of pancreas/duodenum
            - leukaemia
            - lymphoma
            - thymoma
        - drugs
            - antidepressants
            - psychotropics
            - anaesthetic drugs
            - ADH analogues
            - chemotherapy
            - others (e.g. NSAIDs, amiodarone, morphine, PPI, ciprofloxacin)
        - CNS disorders
            - cerebral tumour
            - meningitis/encephalitis
            - brain abscess
            - SAH
            - acute intermittent porphyria
            - SLE
        - hormone deficiency
            - hypothyroidism
            - adrenal insufficiency

        - others
            - Guillain-Barré syndrome
            - HIV
            - hereditary SIADH
            - giant cell arteritis
            - idiopathic
        - pulmonary disorders
            - pneumonia (viral, fungal, bacterial)
            - TB
            - lung abscess
        - management
            - manage hyponatraemia
            - fluid restrict
            - incremental increase in sodium to avoid central pontine myelinolysis
            - medications to decrease ADH secretion
                - demeclocycline
                - tolvaptan
- adrenocortical insufficiency
    - usually secondary adrenal insufficiency (caused by steroid use)
    - causes of crisis
        - major or minor infections (usually GI upset)
        - injury
        - surgery
        - allergy/migraine
        - pregnancy
        - over-exertion/dehydration
        - bereavement/emotional distress
        - acute hypoglycaemia in people with diabetes
        - abrupt withdrawal of steroids
    - symptoms
        - malaise
        - fatigue
        - nausea/vomiting
        - abdominal pain
        - low grade fever
        - muscle pains and cramps
        - hypotension/hypovolaemic shock
        - confusion, loss of consciousness and coma
    - investigations
        - sodium usually moderately decreased but may be normal
        - potassium usually slightly increased or normal
        - creatinine may be raised
        - hypoglycaemia (possibly severe) is characteristic in children
        - calcium may be slightly raised
        - blood for cortisol and ADH should be taken but treatment should be started without waiting for results
    - management

        - IV or IM hydrocortisone:
            - 100mg in an adult
            - 50-100mg for a child aged 6 or more
            - 50mg for a child aged 1-5
            - 25mg for a child under 1
        - fludrocortisone is not required as hydrocortisone has mineralocorticoid activity at these doses
        - rehydration with saline
        - continuous cardiac and frequent electrolyte monitoring
        - after rehydration, 100-200mg hydrocortisone in 5% glucose IV over 24 hours
        - treatment of the underlying precipitating disorder
        - once stabilised, gradual reduction of IV steroid dose and reintroduction of oral therapy
    - prevention
        - steroids should not be stopped suddenly if used for more than 2 weeks
        - good education of patients regarding early dose adjustment (e.g. doubling of dose) when unwell
        - medical emergency bracelet and steroid card
        - emergency self-injection kit and instruction on how to use it

OPHTHALMOLOGY



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OptP1 diplopia

- binocular double vision (89%)
    - images produced by the two eyes do not match and are misaligned

    - diplopia disappears when one eye covered
- monocular double vision
    - much less common
    - continues when the unaffected eye is covered
    - can be caused by abnormalities of the lens, cornea or retina, resulting in splitting of the image
- causes (by anatomical site):
    - cornea and lens
        - refractive errors
        - keratoconus
        - cataracts
        - corneal scarring
        - subluxation
        - herpes zoster
    - eye muscle disorders
        - primary disorders of muscle
            - e.g. myasthenia gravis, Graves' disease, myotonic dystrophy
        - muscle trapping (inferior rectus) due to basal orbital fracture
        - convergence insufficiency (inability to align the eyes when focusing on near objects)
            - worse when tired, treated with glasses, eye exercises or prisms
    - nerve problems
        - problems affecting cranial nerves III, IV, VI
            - includes MS, Guillain-Barré, diabetes
        - temporary palsy of a singular ocular nerve can occur and the cause may be unknown
            - can be associated with infections (e.g. Lyme disease) or inflammatory conditions (e.g. giant cell arteritis)
        - most common with vasculopathies such as diabetes and hypertension
    - nerves can be trapped after orbital bone fracture
    - brain problems
        - vascular conditions
            - stroke, aneurysm, space occupying lesion, migraine, raised ICP
                - raised ICP disproportionately affects VI (abducens)
        - temporary causes
            - alcohol, concussion
        - drug side effect
            - e.g. phenytoin, lamotrigine, opioids, ketamine
- examination:
    - ptosis
    - pupil reaction to light and accommodation
    - pupil abnormalities
    - visual acuity
    - fundoscopy
- red flags:
    - pupil involvement with third nerve palsy

        - large poorly reactive pupil with diplopia is the most common presentation of a posterior communicating artery aneurysm
    - diplopia with two or more of lip, pupil and eye movement
        - may suggest isolated third nerve palsy, Horner's syndrome due to carotid dissection or inflammatory neuropathy (e.g. GBS)
    - multiple cranial nerve palsies
        - suggests intracranial or meningeal tumour, polyneuropathy or cavernous sinus lesion
    - diplopia with weakness or fatigue
        - suggests myasthenia gravis
    - diplopia with new onset headache and scalp tenderness
        - suggests temporal arteritis
- people with diplopia must not drive until diplopia corrected (e.g. with glasses, eye patch)

OptP2 eye trauma including foreign bodies

- corneal trauma
    - usually due to foreign bodies blown or flicked into the eye
        - may become lodged on the cornea or under the upper eyelid
        - foreign bodies travelling at speed may penetrate the orbit – intraocular injury should be considered
    - investigation:
        - thorough eye examination
        - upper lid eversion to look for subtarsal foreign body
        - fluorescein to assess for corneal abrasion or foreign body
        - careful inspection with slit lamp to assess for subtle penetrating foreign body
            - other signs include reduced visual acuity, pupil irregularity, hyphaema and/or vitreous haemorrhage
    - management (non-penetrating corneal trauma):
        - foreign body removal
            - local anaesthetic drops and removal with cotton bud if possible
            - 23G needle can be tried as second line with slit lamp
            - referral to ophthalmology if removal incomplete
        - topical antibiotics
        - dilation of pupil with cyclopentate can ease pain from iris spasm
        - oral analgesics
        - eye pad (only for patient comfort)
        - advise patient not to drive until vision returns to normal
    - management (penetrating corneal trauma):
        - orbital x-ray (eyes up, eyes down) to look for radio-opaque foreign body
        - apply eye shield (not pad to avoid pressure)
        - analgesia
        - tetanus prophylaxis if needed
        - urgent ophthalmology referral
- ocular burns
    - alkali burns are more severe than acid due to deeper penetration
    - check pH of both eyes

    - copious irrigation with 0.9% sodium chloride
        - may need local anaesthetic drops to facilitate
        - continue until pH normal (7.4)
    - after irrigation, examine with slit lamp and fluorescein staining
    - if pH not returning to normal or large burn visible on cornea, refer to ophthalmology

OptP3 painful eye

- ocular
    - acute angle glaucoma (usually red eye)
    - corneal infection, abrasion, foreign body
    - intraocular (choroidal melanoma) and metastatic tumour
    - optic neuropathy
    - dry eye
- orbital
    - orbital infection, inflammation and tumour invasion
- cranial
    - cavernous sinus thrombosis
    - paranasal sinusitis
- neurological
    - cluster headache
    - migraine
    - trigeminal neuralgia
    - elevated intracranial pressure (exacerbated by Valsalva manoeuvre)
- vascular
    - rarely, intracranial bleed or stroke
    - carotid artery disease
    - temporal arteritis

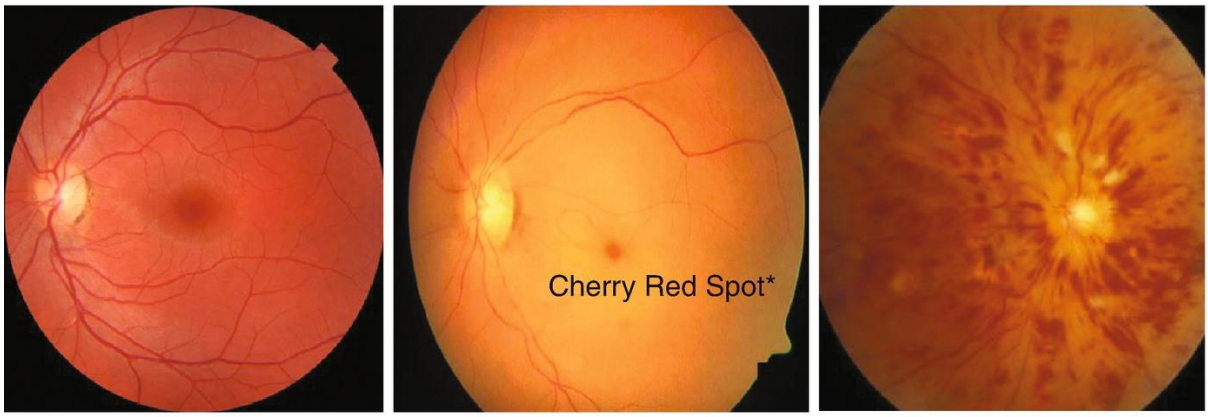
OptP4 red eye

- differential of acute red eye:
    - conjunctivitis
    - corneal abrasion
    - foreign body
    - ocular burn
    - corneal ulcer
    - keratitis
    - acute angle-closure glaucoma
    - anterior uveitis (iritis)
    - scleritis/episcleritis
    - cellulitis
    - subconjunctival haemorrhage
- conjunctivitis
    - bacterial, viral or allergic
    - bacterial causes are most commonly Strep. pneumoniae, H. influenzae, Staph. aureus
        - consider Chlamydia and Gonococcus in adults with urogenital symptoms, neonates and those not responding to treatment

- viral cause is usually adenovirus
    - highly contagious, may affect several family members
- allergic is usually seasonal and associated with other atopic diseases
    - treat with oral or topical antihistamines

OptP5 sudden visual loss

- classification
    - transient v permanent
        - transient
            - TIA
            - migraine
            - vitreous haemorrhage
        - permanent
            - retinal detachment
            - retinal vascular occlusions (untreated)
            - drug related (methanol or quinine poisoning)
            - stroke
    - painless v painful
        - painless
            - central retinal artery occlusion
            - central retinal vein occlusion
            - retinal detachment
        - painful
            - optic neuritis
            - temporal arteritis
            - glaucoma
            - migraine
    - monocular v binocular
        - monocular
            - central retinal artery occlusion
            - central retinal vein occlusion
            - retinal detachment
            - vitreous haemorrhage
        - binocular
            - migraine
            - papilloedema
            - TIA/stroke



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- central retinal artery occlusion (CRAO)
    - end artery, occlusion is usually embolic
    - features:
        - direct pupillary reaction sluggish or absent
            - consensual response intact
        - fundoscopy shows pale retina with cherry red spot at fovea (where choroidal circulation, which is normal, is visible due to thinness of retina)
    - management:
        - re-breathe CO2
            - to vasodilate retinal artery
        - timolol eye drops
            - to reduce intraocular pressure
        - acetazolamide 500mg IV
            - to reduce proportion of aqueous humor and intraocular pressure
        - massage globe
            - aim to dislodge embolus
        - sublingual GTN
            - to vasodilate central retinal artery
- central retinal vein occlusion
    - more common that CRAO
    - presentation:
        - severely decreased visual acuity
        - afferent pupillary defect
        - fundoscopy shows 'stormy sunset' appearance
            - tortuous dilated veins
            - flame haemorrhages
            - hyperaemia
            - cotton-wool spots
    - treatment:
        - management of risk factors to try and preserve other eye
            - risk factors include old age, glaucoma, atherosclerosis, hypertension, polychythaemia

- vitreous haemorrhage and retinal tears
    - vitreous body represents 80% of the eye and is 99% water, 1% hyaluronic acid/collagen
        - fills the space between the lens and the retina
        - adherent to the retina in three places: anteriorly at the border of the retina, at the macula and at the optic nerve
    - with increasing age, vitreous may liquefy and collagen fibres clump together causing the vitreous to collapse
        - pockets left by collapse are seen as floaters
    - vitreous haemorrhage can occur due to the rupture of abnormal blood vessels or due to stress on normal vessels
        - abnormal vessels
            - typically the results of neovascularisation due to ischaemia
                - usually from diabetic retinopathy or coagulopathies (e.g. sickle cell anaemia)
            - new vessels are fragile and prone to rupture
        - normal vessels
            - an rupture when sufficient mechanical force applied
            - if vitreous detaches posteriorly it can pull on and rupture the vessels
                - the force may also cause the retina to tear or detach
            - blunt or penetrating trauma can damage intact vessels (leading cause in patients <40)
    - clinical features:
        - in early or mild haemorrhage:
            - floaters, cobwebs, haze, shadows, red hue
        - severely reduced visual acuity in in large bleeds
        - loss of red reflex
        - retina difficult to visualise on fundoscopy
    - management:
        - sit head up to allow blood to collect inferiorly
        - refer to ophthalmology
            - urgent assessment required to assess for associated retinal tear
- retinal detachment
    - ophthalmological emergency
    - most common in elderly, diabetics, myopes and following trauma
    - more common in severe myopes due to longer eyes causing retina to stretch thinly
    - in diabetics, neovascularisation can cause tractional retinal detachment
    - features:
        - often premonitory flashing lights and increased floaters
            - then curtain develops across vision
        - loss of red reflex on examination
        - retina may be difficult to visualise or appear dark and opalescent
        - visual acuity may be reduced if there is macular involvement, or there may be visual defect
    - refer for urgent surgery and re-attachment
- optic neuritis
    - inflammation of the optic nerve

    - may cause partial or complete visual loss
    - presents sub acutely over a few days
    - features:
        - ache behind the eye
        - pain on eye movements
        - red desaturation (red colour appears less vivid)
        - relevant afferent pupillary defect
        - optic disc may be swollen
    - most recover without treatment
        - there is an association with multiple sclerosis and is the initial presenting symptom in 20-30%
- amaurosis fugax
    - transient monocular visual loss
    - sudden, lasts 5-20 minutes
    - caused by temporary arterial obstruction
        - can be due to different causes:
            - embolic
            - atheromatous disease
            - temporal arteritis
            - acute angle-closure glaucoma
            - increased intracranial pressure
            - hypercoagulability
    - if due to embolus it indicates significant stroke risk and requires immediate investigation

OptC1 acute glaucoma

- pathophysiology:
    - drainage of aqueous humor is compromised, intraocular pressure increases
    - aqueous humor is produced by the ciliary body
        - fills the anterior and posterior chambers
        - passes through the pupil from the posterior into the anterior chamber
        - drained through the trabecular meshwork at the irido-corneal angle into the canal of Schlemm
    - three factors in development of acute angle-closure glaucoma:
        - shallow anterior chamber
            - lens is relatively anterior in the eye
            - associated with hypermetropia (long-sightedness)
        - lens growth
            - lens thickens throughout life and encroaches on anterior chamber
        - pupillary dilatation
            - physiological (low light) or pharmacological (sympathomimetics, anticholinergics)
            - blocks flow of aqueous humor
                - iris contracts and is pushed anteriorly against the trabecular meshwork
                - aqueous reabsorption is impeded
                - intraocular pressure rises

- clinical features:
    - blurred vision/halo around bright lights
        - due to corneal oedema
    - headache
    - eye pain
    - abdominal pain
    - nausea and vomiting
    - decreased visual acuity
    - mid-dilated and unreactive pupil
    - hazy oedematous cornea
    - circumcorneal erythema
    - raised intraocular pressure
- management:
    - high level of suspicion as may appear to be systemic problem
    - pilocarpine 2% eye drops
        - every 5-15 minutes
        - constricts the pupil, opening the angle and improving aqueous humor drainage
        - prophylactic drops to other eye
    - analgesia
        - IV morphine and antiemetic
    - acetazolamide
        - 500mg IV then 500mg PO
        - carbonic anhydrase inhibitor
        - reduces aqueous humor production, reducing intra-ocular pressure
        - weak diuretic
    - mannitol 20%
        - up to 500ml
        - osmotic diuresis which reduces intra-ocular pressure
    - timolol eye drops
        - reduces aqueous humor production
    - urgent ophthalmology referral
    - definitive treatment is laser iridotomy or iridectomy

OptC2 cranial nerve palsy

- systematic approach:
    - muscular (e.g. dystrophy)
    - neuromuscular junction (e.g. myasthenia)
    - cranial nerve lesion outside the brainstem (e.g. compression)
    - cranial nerve lesion within the brainstem (e.g. MS)
- I (olfactory)
    - anatomy:
        - bipolar neurones which pass through the cribiform plate to the olfactory bulb
    - test:
        - block one nostril, ask patient if they can smell something (e.g. alcohol swab, coffee, lavender)

- signs:
    - reduced taste and smell, but not to ammonia (stimulates pain fibres in trigeminal)
- causes:
    - trauma
    - frontal lobe tumour
    - meningitis

- II (optic)
    - anatomy:
        - axons of the retinal ganglion cells
        - at the optic chiasm, the fibres from the nasal parts of the retina decussate, join with the non-decussating fibres and pass backwards in the tracts to the visual cortex
    - test:
        - gross assessment of acuity (read something with each eye)
            - or Snellen chart if available (should wear usual glasses)
        - colour vision with Ishihara plates
        - visual fields
        - fundoscopy
        - pupillary reflex
    - causes:
        - visual field defects
            - monocular
                - lesions of one eye or optic nerve, e.g. MS, giant cell arteritis
            - bilateral
                - methyl alcohol, tobacco amblyopia, neurosyphilis
            - bitemporal hemianopia
                - optic chiasm compression, e.g. internal carotid artery aneurysm, pituitary adenoma, craniopharyngioma
            - homonymous hemianopia (loss of same half of visual field)
                - lesions on the opposite side to the defect, behind the optic chiasm (optic tract, lateral geniculate nucleus, optic radiations or occipital cortex) – e.g. stroke, abscess, tumour
        - pupillary abnormalities
            - anisocoria (unequal pupils)
                - physiological in 20%
                - can suggest Horner's syndrome (e.g. due to carotid dissection) or third nerve palsy due to aneurysm
            - unilateral large pupil (poor constriction in well lit room)
                - traumatic iris damage
                - 3rd nerve palsy
                - rubeosis iridis
                - Holmes-Adie pupil
                    - benign, most commonly affects women in 3rd/4th decade
                    - slow response to light, brisk to accommodation

        - tonic pupil (remains small for a long time after constricting)
        - 80% unilateral
        - due to damage to ciliary ganglion or postganglionic parasympathetic fibres, usually by bacterial or viral infection
        - may need corrective glasses
        - if present with diminished deep tendon reflexes (+/- autonomic nerve dysfunction) is Holmes-Adie syndrome
    - pharmacological dilation
- unilateral small pupil (poor dilatation in low light)
    - physiologically small pupil
    - uveitis with synaechiae
    - Horner's syndrome
        - interruption of sympathetic nerve supply to eye
        - constricted pupil, ptosis, anhidrosis, enophthalmos
        - causes include benign (migraine, goitre, cluster headache), neurological (MS, syringomyelia), compressive (Pancoast's tumour, thyroid carcinoma, cavernous sinus thrombosis, carotid artery dissection
    - Argyll Robertson pupil
        - tonically small pupil with brisk response to accommodation
        - most common cause is neurosyphilis
        - other rare causes include diabetic neuropathy, alcoholic midbrain degeneration, pineal gland tumour, encephalitis, amyloidosis, MS, midbrain tumour
    - pharmacological constriction
- relative afferent pupil defect (defect in direct pupillary response)
    - unilateral optic neuropathy (arteritic or non-arteritic)
    - optic neuritis
    - severe glaucoma
    - traumatic optic neuropathy (direct ocular trauma, orbital trauma, head injury damaging optic nerve)
    - optic nerve tumour
    - orbital disease (e.g. compressive damage to optic nerve from thyroid-related orbitopathy, orbital tumours, vascular malformations)
    - optic atrophy
    - optic nerve infection or inflammation (e.g. cryptococcus, sarcoidosis, Lyme disease)
    - severe ischaemic retinal disease (e.g. ischaemia central retinal vein occlusion, central retinal artery occlusion, sickle-cell retinopathy)
    - retinal detachment

        - very severe unilateral macular degeneration
        - retinal infection (e.g. CMV, herpes simplex)
        - amblyopia
    - non-reactive pupil (suggests injury or compression of 3rd nerve and upper brain stem)
        - unilateral non-reactive pupil
            - post-traumatic iridocyclitis (e.g. direct facial trauma)
            - serious intracranial pathology (e.g. intracranial mass, haemorrhage)
            - diffuse brain injury
            - oculomotor (III) nerve palsy
            - posterior communicating artery aneurysm (large poorly reactive pupil with diplopia)
            - pharmacological blockade
            - ocular prosthesis
        - bilateral non-reactive pupils
            - extensive intracranial pathology
            - diffuse brain injury
            - brain stem herniation/brain death
            - pharmacological blockade
- optic neuritis (pain of moving eye, loss of central vision, afferent pupillary defect, papilloedema)
    - demyelination
    - rarely: sinusitis, syphilis, collagen vascular disorders
- optic atrophy (pale discs and reduced acuity)
    - MS, frontal tumours, Friedrich's ataxia, retinitis pigmentosa, syphilis, glaucoma, Leber's optic atrophy, optic nerve compression
- papilloedema
    - raised intracranial pressure: tumour, abscess, encephalitis, hydrocephalus, idiopathic intracranial hypertension
    - retro-orbital lesion (e.g. cavernous sinus thrombosis)
    - inflammation (e.g. optic neuritis)
    - ischaemia (e.g. accelerated hypertension)

- III (oculomotor)
    - anatomy:
        - emerges from midbrain on medial aspect of crus cerebri. passes forward between posterior cerebral and superior cerebellar arteries very close to posterior communicating artery
        - pierces dura near edge of tentorium cerebelli
        - passes through lateral part of cavernous sinus with IV and V
    - test:
        - (with IV and VI) accommodation and eye movements
    - signs:
        - fixed dilated pupil which does not accommodate
        - ptosis
        - complete internal ophthalmoplegia (masked by ptosis)
        - outward deviation due to unopposed lateral rectus

- causes:
    - myasthenia gravis
    - thyroid eye disease
    - chronic progressive external ophthalmoplegia
    - orbital inflammatory pseudotumour
    - internuclear ophthalmoplegia
    - Parinaud's syndrome (vertical gaze palsy caused by pineal tumour)
    - giant cell arteritis
    - extradural haematoma
    - intracranial or meningeal tumour, polyneuropathy or cavernous sinus lesion (with other palsies)

- IV (trochlear)
    - anatomy:
        - passes backward in the brainstem, decussates in anterior medullary vellum and emerges to go around the cerebral peduncle, passing over the tentorium
        - enters the cavernous sinus with II and VI
        - enters the orbit to supply the superior oblique muscle
    - signs:
        - diplopia due to weakness of downward/inward movement
        - most common cause of pure vertical diplopia
            - patient compensates by tilting head away from affected side
    - causes:
        - rare as isolated lesion, usually due to orbital trauma
        - may occur in diabetes of infarction secondary to hypertension
- V (trigeminal)
    - anatomy:
        - 3 divisions: ophthalmic, maxillary and mandibular
            - ophthalmic supplies skin over medial nose, forehead and eye
                - including corneal reflex
            - maxillary
                - supplies skin of upper lip, cheek, triangle of skin from angle of eye and mouth to apex in mid-temporal region
            - mandibular
                - skin from lower lip and chin up to and including tragus and upper part of pinna
                - mucous membranes of floor of mouth, cheek, anterior two thirds of tongue
                - motor fibres supply masseter, temporalis and pterygoids
    - test:
        - compare touch at ophthalmic, maxillary and mandibular branches
        - clench teeth to check masseters
        - corneal reflex (motor VII)
    - signs:
        - reduced sensation over affected area
        - weakness of jaw clenching and side to side movement
        - jaw deviates to side of LMN lesion when mouth open

        - fasciculation of temporalis and masseter
    - causes:
        - sensory
            - trigeminal neuralgia
            - herpes zoster
            - nasopharyngeal carcinoma
        - motor
            - bulbar palsy
            - acoustic neuroma
- VI (abducens)
    - anatomy:
        - from nucleus in floor of fourth ventricle, through pons to follow long extracerebral course on base of brain; through posterior fossa near dorsum sellae to cavernous sinus then orbit and lateral rectus
    - signs:
        - inability to look laterally
        - eye is deviated medially due to unopposed action of medial rectus
    - causes:
        - MS
        - pontine CVA
- VII (facial)
    - anatomy:
        - mainly motor (some sensory fibres from external acoustic meatus, fibres controlling salivation and taste fibres from anterior tongue)
        - fibres pass through the internal auditory meatus, through the petrous temporal in the facial canal
            - widens to form the geniculate ganglion (taste and salivation) on medial side of middle ear then turns sharply (and chorda tympani leaves) to emerge through stylomastoid foramen
    - test:
        - furrow brow/raise eyebrows
        - close eyes and hold closed
        - puff out cheeks
        - smile
    - causes:
        - LMN
            - Bell's palsy
            - polio
            - otitis media
            - skull fracture
            - cerebellopontine fracture
            - parotid tumour
            - herpes zoster (Ramsay Hunt syndrome)
            - Lyme disease
        - UMN (spares forehead – bilateral innervation)
            - stroke
            - tumour

- VIII (vestibulocochlear)
    - anatomy:
        - two group of fibres to cochlea (hearing) and to semicircular canals, utricle and saccule (balance and posture)
        - pass with facial nerve from brainstem across posterior fossa to internal acoustic meatus
    - test:
        - whisper or use high frequency (516Hz) tuning fork
            - with tuning fork compare air to bone conduction
            - Weber's test if significant loss in one ear – tuning fork in centre of forehead will be quieter in bad ear with nerve deafness and louder with conductive deafness
    - signs:
        - unilateral sensorineural deafness, tinnitus
    - causes:
        - loud noise
        - Paget's disease of home
        - Ménière's disease
        - herpes zoster
        - neurofibroma
        - acoustic neuroma
        - brainstem CVA
        - lead
        - aminoglycosides
        - furosemide
        - aspirin
- IX (glossopharyngeal)
    - anatomy:
        - sensory, motor (stylopharyngeus) and parasympathetic (salivary gland) fibres
        - across posterior fossa, through jugular foramen into neck
        - supplies tonsil, palate and posterior third of tongue
    - test:
        - can check taste to posterior third of tongue but not usually done
    - signs:
        - only with bilateral lesions
            - pseudobulbar palsy
    - causes:
        - trauma
        - brainstem lesions
        - cerebellopontine angle and neck tumours
        - polio
        - Guillain-Barré syndrome
- X (vagus)
    - anatomy:
        - motor (to palate and vocal cords), sensory (posterior and floor of external acoustic meatus), visceral afferent and efferent fibres

        - leaves skull through jugular foramen, passes within carotid sheath in neck (giving off cardiac branches and recurrent laryngeal supplying vocal cords), through thorax (supplying lungs) and through oesophageal opening to supply abdominal organs
    - test:
        - (with IX) swallow, say ah
        - gag reflex if required
    - signs:
        - nasal speech and nasal regurgitation of food due to palatal weakness
        - asymmetrical movement of palate
        - recurrent nerve palsy results in hoarseness, loss of volume and 'bovine' cough
    - causes:
        - trauma
        - brainstem lesions
        - cerebellopontine angle/jugular foramen/neck tumours
        - polio
        - Guillain-Barré syndrome
- XI (spinal accessory)
    - anatomy:
        - motor to sternocleidomastoid and trapezius
    - test:
        - shrug shoulders against resistance, push head against hand
    - signs:
        - weakness and muscle wasting
    - causes:
        - as vagus nerve
- XII (hypoglossal)
    - anatomy:
        - passes briefly across posterior fossa, leaves skull through hypoglossal canal
        - supplies motor fibres to tongue and most of infrahyoid muscles
    - test:
        - stick tongue out, look for deviation/fasciculation
    - signs:
        - wasting of ipsilateral side of tongue with fasciculation
        - deviation of tongue towards affected side on protrusion for LMN lesion, deviates away from side of central lesion
    - causes:
        - polio
        - syringomyelia
        - tuberculosis
        - median branch thrombosis of vertebral artery

OptC3 orbital/preseptal and peri-orbital cellulitis

- orbital cellulitis
    - pathophysiology:
        - infection of tissues posterior to orbital septum

                - orbital septum extends from orbital rims to the eyelids
            - may arise from direct extension from peri-orbital structures, direct inoculation of the eye from trauma/surgery or via haematogenous spread
            - most common organisms are Strep. pneumoniae, Strep. pyogenes, Staph. aureus, H. influenzae
    - signs:
        - loss of vision/red de-saturation
            - due to optic nerve compression
        - ophthalmoplegia
        - painful eye movements
        - proptosis
        - chemosis
        - conjunctival oedema
    - management:
        - broad-spectrum IV antibiotics
        - urgent ophthalmology referral
        - CT scan to look for abscess formation
        - may require surgical drainage if visual acuity reduces, afferent pupillary defect develops or antibiotics fail to improve symptoms
    - complications:
        - visual loss
        - death
        - septicaemia
        - cavernous sinus thrombosis
        - central retinal artery occlusion
        - secondary glaucoma
        - optic neuritis
        - osteomyelitis
        - meningitis
        - orbital abscess
        - endophthalmitis
- peri-orbital (preseptal) cellulitis
    - pathophysiology:
        - tends to be less severe than orbital cellulitis
        - involves eyelids and soft tissues anterior to orbital septum
        - usually follows upper respiratory tract infection, external ocular infection or eyelid trauma
        - most common organisms Staph or Strep
    - signs:
        - eyelid and periorbital oedema, erythema, discomfort
        - normal ocular exam
            - normal acuity
            - normal eye movements with no significant discomfort
            - no proptosis
    - management:
        - co-amoxiclav or cephalosporin
            - can be oral

- IV may be required if it is a child, there is inadequate response to initial oral course or concerns that it may be orbital cellulitis

OptC4 ophthalmia neonatorum

- definition and aetiology;
  - any conjunctivitis in first 28 days of life
  - most commonly infective in origin
    - N. gonorrhoeae used to be most common cause, now accounts for <1%
    - Chlamydia trachomatis also on decline
    - 30-50% are non-sexually transmitted bacterial:
      - Staph. aureus
      - Strep. penumoniae
      - Pseudomonas spp.
      - Haemophilus spp.
    - viral infection (less common):
      - herpes simplex
      - adenovirus
      - enterovirus
    - can also be a reaction to chemical irritants
- presentation:
  - purulent, mucopurulent or mucoid discharge from one or both eyes within first month of life
    - typically with injected conjunctiva and lid swelling
    - may have associated systemic infection
  - chemical conjunctivitis:
    - mild irritation, tearing and redness
    - follows prophylactic silver nitrate administration (for gonorrhoeal infection) in past 24-48 hours
  - bacterial conjunctivitis:
    - usually has longer incubation period
      - subacute onset between 4^{th} and 28^{th} day of life
    - may be mixed picture with red eye with lid swelling, varying amount of purulent discharge
    - gonorrhoeal infection:
      - typically 2-5 days after birth (may be later)
      - hyperacute conjunctival injection and chemosis, lid oedema, severe purulent discharge
      - may be corneal ulceration and perforation
    - chlamydial infection:
      - 5-12 days after birth (may be later)
      - unilateral/bilateral watery discharge which becomes copious and purulent
      - may be associated preseptal cellulitis
      - less commonly, rhinitis, otitis, pneumonitis
      - eyes usually less inflamed than in gonorrhoeal infection
  - viral conjunctivitis:
    - acute onset 1-14 days after birth

        - unilateral/bilateral serosanginous discharge +/- vesicular skin lesions
        - other features include:
            - keratitis
            - anterior uveitis
            - cataract
            - retinitis
            - optic neuritis (rarely)
        - uncommonly can have systemic infection:
            - jaundice
            - hepatosplenomegaly
            - pneumonitis
            - meningoencephalitis
            - DIC
- referral:
    - the majority of neonates with sticky discharge have a benign cause, usually blocked nasolacrimal ducts (do not have red eyes)
    - referral for:
        - conjunctival redness (particularly if conjunctiva underlying sclera is involved)
        - sudden and severe onset
        - distressed or unwell baby
        - both eyes affected
        - maternal gonococcal infection suspected
        - maternal concern
- management:
    - chemical conjunctivitis:
        - self-limiting, no treatment required
        - early review at 24 hours to confirm diagnosis
    - bacterial infection:
        - guided by organism grown
        - may require hospitalisation and IV antibiotics
        - infections that are not chlamydia or gonorrhoea may be managed with topical antibiotics
    - viral infections:
        - IV acyclovir for 14 and up to 21 days
- complications:
    - gonococcal
        - keratitis
        - conjunctival scarring
        - superior corneal pannus
        - side effects of treatment
        - permanent visual impairment
    - overwhelming systemic infection
        - e.g. chlamydial pneumonia, disseminated herpes simplex

OptC5 inflammatory eye disease

- keratitis
    - breach of corneal epithelium

- may be due to infection or trauma
- features:
  - corneal ulceration
    - may be punctate, rounded or dendritic
  - unilateral painful, photophobic, injected eye
  - vision may be affected depending on location of ulcer
  - superficial keratitis:
    - arc eye/snow blindness
    - less serious than keratitis
    - occurs following exposure to UV light without adequate eye protection (e.g. welders, skiers, sunbed users)
    - presents with pain, watering and blepharospasm
    - fluorescein shows multiple punctate corneal lesions
- management:
  - all patients with keratitis or corneal ulcers should have urgent ophthalmology referral
  - superficial keratitis should be managed with NSAID eye drops, cyclopentolate, oral analgesia, eye pad
    - usually resolves in 24 hours

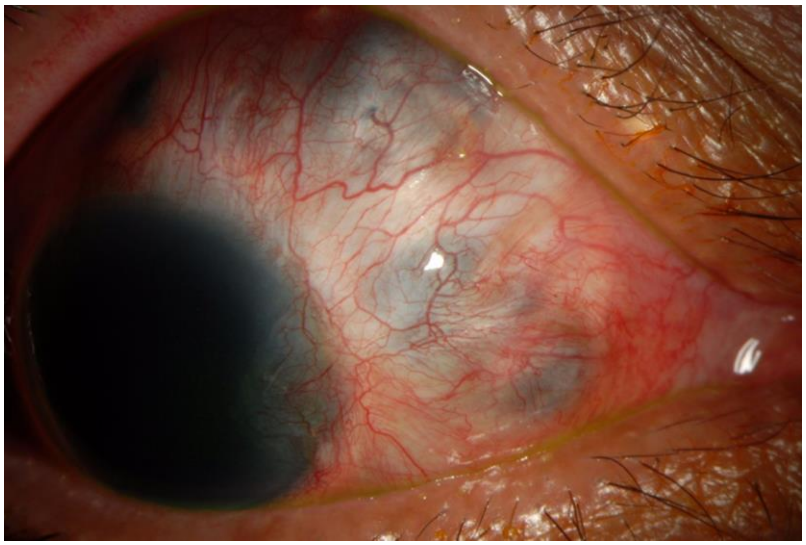
- anterior uveitis (also called iritis)

Credit:ISM / BARRAQUER, Barcelona

- anatomy:
  - uveal tract is made up of choroid, ciliary body and iris
  - choroid forms in the posterior uveal tract
  - the ciliary body and iris form the anterior uveal tract
- associations:
  - autoimmune conditions
    - ankylosing spondylitis
    - ulcerative colitis
    - sarcoid
  - HLA-B27 serotype
  - can also occur following trauma, ocular surgery or keratitis
- presentation:
  - photophobic, painful eye

        - pain described as ‘deep’
        - pain exacerbated by accommodation (due to iris constriction)
    - photophobia may be consensual as light shone in the unaffected eye causes constriction
- signs:
    - circumcorneal erythema
    - irregular pupil due to formation of posterior synechiae (adhesions between iris and lens)
    - slit lamp examination may show inflammatory cells in the anterior chamber and a foggy appearance due to flare from protein leaking from inflamed blood vessels
- management:
    - urgent referral to ophthalmology

- scleritis



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- anatomy:
    - sclera forms a protective cover for the eye
    - avascular
        - receives blood supply from the choroid inside the eye and a deep vascular plexus lying in the episcleral
    - three layers on the outer surface: episcleral, Tenon’s capsule, conjunctiva
- pathophysiology:
    - inflammatory process involving the deep episcleral vascular process
    - association with rheumatological disorders, particularly rheumatoid arthritis and Wegener’s granulomatosis
- clinical features:
    - localised or generalised bluish discoloration of sclera
    - deep dull ache in eye
    - painful eye movements
        - extraocular muscles insert into the sclera
    - visual acuity may be reduced

        - palpation of the orbit is painful
    - urgent ophthalmology referral required
        - inflammation may ultimately lead to thinning of the sclera and perforation of the globe
- episcleritis
    - self-limiting condition involving the superficial episcleral vascular plexus
    - localised inflammation of the superficial plexus
    - patients report irritation rather than pain
    - topical or oral NSAIDs can be used

OptC6 temporal arteritis

- pathophysiology:
    - relatively common vasculopathy affecting patients over 50
    - strongly associated with polymyalgia rheumatica
    - inflammation of the temporal artery results in temporal lobe headaches
    - involvement of the ophthalmic, posterior ciliary and central retinal artery can cause irreversible visual impairment and ischaemic optic neuritis
- clinical features:
    - headache (temporal region)
    - scalp/forehead tenderness
    - jaw claudication
    - relative afferent pupillary defect
    - red de-saturation
    - unilateral visual loss
        - progresses to bilateral in up to 30%
    - pale and swollen optic disc
    - symptoms of PMR
        - malaise, myalgia in shoulders and pelvic region, weight loss
- investigations:
    - ESR, CRP
    - temporal artery biopsy
- management:
    - empirical steroids (start at 60mg prednisolone daily)
    - joint ophthalmology and rheumatology referral

PAIN AND SEDATION

PC1 analgesics

- background
    - pain is the most common presenting complaint to the ED
    - evidence shows that we are not good at relieving pain in a timely fashion
- pathophysiology and psychology of pain
    - tissue damage at a cellular level results in the release of chemicals which stimulate pain receptors

        - these pain receptors produce an electrical impulse which is transmitted via a peripheral verve fibre to the spinal cord and then to the brain, where the impulse is perceived as pain
    - tissue damage may be:
        - mechanical
        - thermal
        - chemical
        - electrical
        - metabolic (e.g. hypoxaemia, hypoglycaemia)
    - prostaglandins, which may be produced as a consequence of the injury, sensitise the pain receptors to the chemicals that are released
    - pain receptors
        - also called nociceptors
        - distributed throughout the body
        - can be thought of as free nerve endings
        - produce an electrical impulse and connect to peripheral nerve fibres
    - peripheral nerve fibres transmit the electrical impulse to the dorsal horns of the spinal cord
    - the ascending tracts within the spinothalamic tract transmit impulses to the brain
    - pain is perceived in the brain
        - influences on the perception of pain include:
            - emotion
            - environment
            - culture
            - education
            - beliefs
            - personality
            - previous experiences
        - some analgesics, distraction techniques and hypnosis act directly on the brain and not at the injury site
    - visceral pain pathways
        - involve the gut, heart, lungs etc.
        - more crude and result in poorer localisation of pain and referred pain
- WHO analgesic ladder (should be entered at appropriate point)
    - step 1 – non-opioid analgesics
        - e.g. aspirin, paracetamol, NSAIDs
        - given regularly and use adjuncts if appropriate
    - step 2 – mild opioids
        - codeine – effective for relief of mild to moderate pain but too constipating for long-term use
        - dihydrocodeine – similar efficacy to codeine, can be given 4 hourly with individual dose adjustment
    - step 3 – strong opioids
        - useful for moderate to severe pain, particularly of visceral origin
        - main side effects are nausea, vomiting, constipation, drowsiness and in larger doses respiratory depression and hypotension; occasionally hyperalgesia occurs

- opiates
    - first choice for moderate to severe pain
    - a downside is variability in patient response
        - frequent reassessment is required
    - morphine
        - RCEM recommends 0.1-0.2mg/kg of IV morphine initially for severe pain
        - reaches a peak within a few minutes when given IV and generally provides clinical effect for 3-4 hours
        - IV or SC administration should not be used given delayed absorption and pain in repeated administration
    - diamorphine
        - no differences between parenteral diamorphine and morphine in terms of analgesia and side effects
        - however, diamorphine can be rapidly absorbed by the transmucosal route
            - it is used intranasally in children and is an option in adults with difficult venous access
    - fentanyl
        - has brief action, providing analgesia for 30-45 minutes
            - therefore has a role in procedural sedation
    - there are other ultra-short acting opiates such as remifentanil (duration <6 minutes) and alfentanil (about 10 minutes)
    - codeine
        - compares poorly with both paracetamol and ibuprofen
        - most of the analgesic effect is as a result of metabolism to morphine
            - nearly 10% of Caucasians and 1-2% of Asians lack the required enzyme
    - tramadol
        - widely used in continental Europe
        - not all of the effects are via opiate receptors
        - maximum dose is 100mg every four hours
        - has a better safety profile in terms of respiratory depression
        - can have psychiatric reactions
        - can cause dependency and withdrawal problems
        - can react with antidepressants such as SSRIs and increase risk of serotonin toxicity
    - pethidine
        - short duration of action
        - less constipating than morphine but less potent
        - not suitable for severe continuing pain (metabolite build up can cause tremor, confusion and convulsions)
    - methadone
        - less sedating than morphine and acts for longer
        - risk of accumulation and overdose if given more than twice a day long-term
    - common adverse effects of opiates
        - sedation
        - respiratory depression
        - nausea and vomiting
        - histamine release causing pruritus and (mild) hypotension

        - slowing of gastrointestinal function and constipation
        - urinary retention
    - adverse effects tend to be dose-related
    - opiates are generally metabolised in the liver and excreted in the kidneys so may accumulate in patients with liver and kidney dysfunction
- paracetamol
    - analgesic and antipyretic
        - mechanism for either is not known
    - oral, rectal and IV preparations available
    - should be avoided in patients with active liver disease
- NSAIDs
    - have the following properties:
        - analgesic
        - antipyretic
        - anti-inflammatory
    - properties result from the inhibition of prostaglandin synthesis
    - available in oral, rectal, IV, IM and topical preparations
    - there is no good evidence than any NSAID is superior
    - side effects include:
        - peptic ulceration
            - for those at risk, the BNF recommends coprescription of a PPI, an H2-receptor antagonist such as ranitidine at twice the usual dose, or misoprostol
        - renal impairment
            - particularly in patients with pre-existing renal impairment or hypotension
        - bronchospasm
            - in a small percentage of asthmatics (typically those with chronic rhinitis and nasal polyps)
        - inhibition of platelet function with some evidence of increased blood loss following surgery
- adjuvant therapy
    - antidepressants
        - low dose antidepressants (e.g. 75-150mg amitriptyline ON) are useful for neuropathic pain
        - SSRIs may also be helpful
    - anticonvulsants
        - can be used for neuropathic pain, but evidence is inconsistent
        - examples include carbamazepine, gabapentin and pregabalin
    - muscle spam
        - muscle relaxants such as diazepam or baclofen could be considered
    - nerve compression
        - may be helped by a corticosteroid such as dexamethasone to reduce oedema
    - NMDA-receptor agonists
        - ketamine and methadone may be helpful in controlling the conduction of pain

- physical methods
    - local anaesthetics
        - block nerve conduction reversibly
        - regional blocks have been used to good effect in shoulder pain, intercostal neuralgia, postoperative scar pains, other peripheral neuralgias
    - epidural steroids and facet joint blocks
        - may be used for chronic back pain
        - work best when used early
        - may take a week to have an effect
        - a course of three injections is often recommended
    - TENS (transcutaneous electrical nerve stimulation)
        - uses gate theory as its rationale
        - effectiveness has been challenged but may be useful in some circumstances
    - irreversible physical methods
        - some patients may be offered destruction of nerves (although plasticity means that the problem may return), surgery (such as fixation of long bone fractures or shunts to drain progressive ascites) or neurosurgical interventions for orthopaedic pain

PC2 non-pharmacological methods of pain management

- acute
    - information
        - accurate information given in a way the patient can understand
    - distraction
        - such as videos and games, books or colouring
    - breathing exercises
        - can be done with blowing bubbles
    - immobilisation
        - for limb injuries
    - environment
        - making the environment as non-threatening as possible, e.g. sensory room
- long-term (usually via GP)
    - acupuncture
        - benefit shown in systematic reviews for certain types of pain such as osteoarthritis
    - chiropractic
        - can be helpful for acute and chronic neck pain and back pain
    - physiotherapy
        - popular and economically viable but limited long-term success demonstrated in systematic reviews
    - behavioural management
        - back schools
        - cognitive behavioural therapy
        - fitness training
        - activity scheduling
        - pain management methods
    - complementary medicine

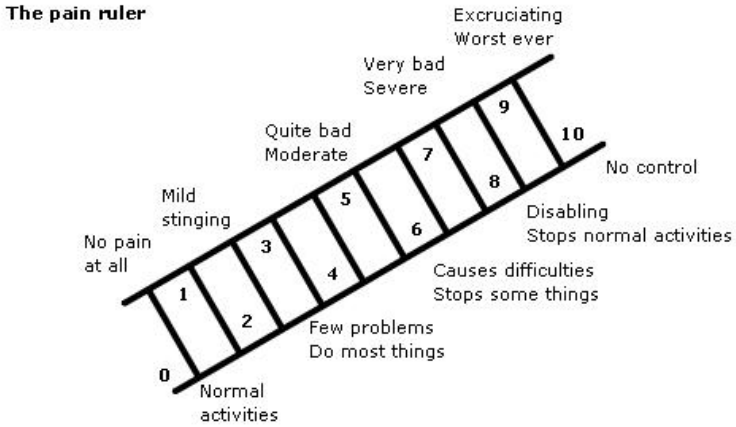
        - found helpful by some patients (e.g. hypnotherapy, herbal treatments, homeopathy)

PC3 pain assessment

- background
    - definition of pain is an unpleasant sensory or emotional experience associated with actual or potential tissue damage
    - intensity of pain is subjective
    - facial expression and demeanour may give some clues but are not reliable
    - physiological parameters (e.g. heart rate, respiratory rate, blood pressure) lack sensitivity and specificity
    - self reporting of pain by patients is to be encouraged
    - pain scoring should be done frequently to assess response
    - there is reasonable evidence that mandatory pain scoring does not necessarily result in adequate analgesic provision
- pain history
    - site
    - duration
    - speed of onset
    - intermittent or constant
    - character of pain
    - aggravating and relieving factors
    - impact on daily living
    - social, emotional and psychological aspects
    - severity
- reasons for using pain tools
    - proper consideration of pain
    - guidance of selection of analgesic agents
    - provide an indication of response to pain and analgesia
        - change in score and trend are more important than the initial score
    - allow departmental audit
    - provide valid methodology for research of pain therapies
        - a VAS change of at least 13mm is required to achieve clinical significance
- verbal descriptor scales
    - simple pain rating
        - no pain
        - mild pain
        - moderate pain
        - severe pain
        - very severe pain
        - worst possible pain
    - rating of pain relief
        - none
        - mild pain relief
        - moderate pain relief
        - complete pain relief
- numerical and combined rating sales

- visual analogue scales
    - 100mm lines with verbal anchors
    - the patient is asked to mark their pain on the horizontal line according to how bad it is, ranging from no pain to pain as bad as it could possibly be
    - the same can be used for pain relief, with a scale from complete pain relief to no pain relief
- verbal numerical rating scale
    - ask the patient to score their pain from 0 to 10 where 0 is no pain and 10 is the worst possible pain
- combined verbal and numerical scale
    - has numbers along the scale but also descriptors at certain points (e.g. agonising, horrible, dreadful, uncomfortable, annoying, none)

- paediatric scales
    - faces scale
    - pain ruler



https://www.rcemlearning.co.uk/reference/pain-management-in-adults/#1570786515027-563ba4c1-fa84

- situations of difficult communication
    - listen to family members and carers
    - nursing opinion
    - physiological clues such as increased heart rate and BP
    - appropriate tools:
        - interpreter
        - multilingual printed information
        - pain measurement scales
        - sign language interpreter
        - dedicated learning needs nurse
        - writing board
    - titration of analgesia
- issues to consider in the Emergency Department
    - are patients encouraged to report pain?

    - what pain assessment tools are in use?
    - when and where are pain assessment tools applied?
    - where is the pain score recorded? is this ideal?
    - are the pain assessment tools appropriate for the ED case mix?
    - when was the last time your department audited its performance regarding analgesic provision?
    - what did the audit show?
    - what changes were suggested and implemented?
    - was change effective?
- strategies for addressing pain
    - musculoskeletal pain (severe)
        - titrated intravenous morphine
            - no requirement for prophylactic antiemetics as there is low incidence of opiate-related nausea (<4%) in patients with musculoskeletal injury
        - if any dislocation, prompt reduction with procedural sedation
        - correction of any significant angulation of long bones with splinting (usually using nitrous oxide)
        - regional anaesthesia in specific injuries:
            - 3 in 1 block (femoral nerve, obturator nerve and lateral cutaneous nerve of the thigh) or fascia iliaca block
                - for fractured femur
                - no evidence of delayed diagnosis of compartment syndrome as a result
            - thoracic epidural
                - for fractured ribs, particularly in patients with chronic lung disease
        - paracetamol may reduce overall opiate requirements
        - NSAIDs are best avoided if ORIF is planned and/or in the elderly or hypotensive/hypovolaemic patient
        - compartment syndrome should be considered if there is pain out of proportion
    - musculoskeletal pain (mild to moderate)
        - paracetamol +/- ibuprofen
        - top up with opiates as required (e.g. tramadol)
        - physical aids:
            - ankle strapping
            - compression support (Tubigrip) for knees
            - slings and splints for upper limb injuries
        - early mobilisation, especially for neck, back and shoulder soft tissue injuries
        - calcitonin is effective in the treatment of acute pain after osteoporosis-related vertebral fractures
    - chest pain
        - goals:
            - optimise oxygen delivery
            - decrease myocardial consumption
            - restore coronary blood flow

        - combination of high flow oxygen, nitrates and morphine with early PCI if STEMI
        - nitrates
            - if no relief from 3x sublingual move to titrated IV
            - usual starting rate is 2-4ml/hr of 50mg in 50ml
                - increase every 2 minutes to effect (it is a low dose compared to the sublingual dose)
            - contraindicated in patients relying on adequate venous return – e.g. aortic stenosis or right ventricular infarct
        - hypotension
            - recognised side effect of nitrates, morphine and perhaps thrombolytic
            - leg elevation and fluid boluses should be considered before stopping nitrate
        - antiemetic
            - should be considered prophylactically, avoiding cyclizine which can increase heart rate and therefore myocardial oxygen consumption
        - nitrous oxide has a proven role
        - for STEMI patients, beta blockers (metoprolol 50mg) or calcium channel blockers (diltiazem 60mg) can be considered, particularly if heart rate or BP remain high
    - cocaine-associated chest pain
        - IV benzodiazepines and nitrates titrated to effect
    - abdominal pain
        - titrated IV morphine
        - IV paracetamol might reduce opiate requirements
        - NSAIDs such as IV ketorolac are as effective as parenteral opiates for biliary colic and more effective than Buscopan
            - they should be avoided in undifferentiated RUQ/epigastric pain in case of peptic ulceration
        - antispasmodics such as buscopan or peppermint oil may be useful for irritable bowel syndrome
    - burns
        - immediate cooling limits damage and provides relief
        - depth and size should be assessed promptly and the burn then covered which has an analgesic effect (e.g. clingfilm, Jelonet)
        - titrated IV morphine alongside paracetamol and ibuprofen
    - sickle cell crisis
        - titrated IV morphine, ideally followed by patient controlled analgesia
        - oxygen and ketorolac have not been shown to be helpful
    - migraine
        - triptans
            - ideally subcutaneous or nasal for faster symptom relief and higher efficacy, particularly with nausea and vomiting
            - oral triptans are better tolerated but take longer to work and are less reliable
        - other effective treatments in the ED include:

                - parenteral metoclopramide
                - chlorpromazine
                - prochlorperazine
            - opiates are not recommended
    - cluster headaches
        - SC sumatriptan or IN sumatriptan can be effective
        - oxygen therapy, 7-10 litres/min for 15 minutes
    - pain out of proportion
        - consider:
            - compartment syndrome
            - Lisfranc injury
            - necrotising fasciitis

PC4 sedation

- background
    - aim is to make the procedural experience as comfortable as possible for the patient whilst ensuring safe practice
    - sedation is a continuum from normal conscious level to fully unresponsive
        - it is difficult to maintain a patient at a pre-defined target level
    - American Society of Anaesthesiologists (ASA) definitions:
        - minimal sedation (anxiolysis)
            - a drug-induced state where patients respond normally to verbal commands
            - cognitive function and coordination may be impaired but ventilatory and cardiovascular function are unaffected
        - moderate sedation (conscious sedation)
            - drug induced depression of consciousness during which patients respond purposefully to verbal commands, either alone or accompanied by light tactile stimulation
            - no interventions are required to maintain a patent airway and spontaneous ventilation is adequate
            - cardiovascular function is usually maintained
        - deep sedation
            - drug induced depression of consciousness during which the patient is not rousable, even by painful stimulation
            - the ability to independently maintain ventilatory function is often impaired
            - the patient requires assistance in maintaining an airway, and positive pressure ventilation may be required because of depressed spontaneous ventilation or drug-induced depression of neuromuscular function
            - cardiovascular function may be impaired
        - dissociative sedation is not included in the classification – a trance-like cataleptic state produced by ketamine and characterised by profound analgesia and amnesia, with retention of protective airway reflexes, spontaneous respirations and cardiopulmonary stability
    - the sedation continuum

        - alert – anxious
        - alert – calm (anxiolysis)
        - drowsy but clear mentation (sedation)
        - eyes open, speech slurred
        - eyes closed but answers questions appropriately
        - opens eyes to voice; is confused
        - oxygen desaturation on room air
        - opens eyes to pain; responds purposefully
        - eyes closed; moans and withdraws from pain
        - moans to pain; non-specific motor response to pain
        - CO_2 retention
        - oxygen desaturation on 2L O_2
        - no response to pain
        - bradycardia, poor gag reflex
        - apnoea, hypotension
        - death
    - ASA physical status classification
        - class I
            - normal, healthy patient
        - class II
            - mild systemic disease
            - e.g. asthma, controlled diabetes
        - class III
            - moderate systemic disease
            - e.g. stable angina, diabetes with hyperglycaemia, moderate COPD
        - class IV
            - severe systemic disease
            - e.g. unstable angina, DKA
        - class V
            - moribund
            - not expected to survive without an operation
        - class VI
            - declared brain-dead
            - patient whose organs are being removed for donor purposes
- considerations before proceeding
    - whether analgesic needs have been appropriately met with opioids/paracetamol/ibuprofen and/or local or regional anaesthesia; whether the procedure could be performed with local anaesthetic and nitrous oxide
    - whether an empathetic approach and clear explanation of the procedure have been provided, alongside distraction such as a conversation, music or a tablet computer
    - whether a general anaesthetic is more appropriate or the procedure is more complicated than thought
    - whether current workload, staffing and environment allows for safe sedation and whether performing procedural sedation now compromises the safety or quality of care for other patients
- typical indications for procedural sedation
    - emergent

        - cardioversion for life-threatening dysrhythmia
        - reduction of markedly angulated fracture/dislocation with soft tissue or vascular compromise
        - intractable pain or suffering
    - urgent
        - care of dirty wounds and lacerations, animal or human bites
        - fracture reduction
        - shoulder reduction
        - hip reduction
        - arthrocentesis
        - neuroimaging for trauma
    - semi-urgent
        - care of clean wounds and lacerations
        - foreign body removal
        - sexual assault examination
- pre-sedation
    - it is important to identify patients who
        - may predictably be difficult to ventilate
        - may predictably desaturate
        - are more likely to regurgitate and potentially aspirate
        - might predictably drop their blood pressure
    - airway assessment (caution if more than one factor present)
        - history
            - previous problems with anaesthesia or sedation
                - check hospital and ED records if possible
            - stridor, snoring or sleep apnoea
            - advanced rheumatoid arthritis
            - chromosomal abnormality (e.g. trisomy 21)
        - examination
            - habitus
                - significant obesity (especially involving neck and face)
            - head and neck
                - short neck
                - limited neck extension
                - decreased hyoid-mental distance (<3cm in an adult)
                - neck mass
                - cervical spine disease or trauma
                - tracheal deviation
                - dysmorphic facial features (e.g. Pierre Robin syndrome)
                - excessive facial hair
            - mouth
                - small opening (<3cm in an adult)
                - edentulous
                - protruding incisors
                - high arched palate
                - macroglossia
                - tonsillar hypertrophy

        - nonvisible uvula
    - jaw
        - micrognathia
        - retrognathia
        - trismus
        - significant malocclusion
- mnemonic BOOTS
    - **b**eard
    - **o**bese
    - **o**lder patient
    - **t**oothless
    - **s**nores
- fasting
    - not required for minimal sedation, sedation with Entonox alone or moderate sedation where verbal contact is maintained
    - guidance for general anaesthesia is 2 hours for clear fluids and 6 hours for everything else
    - in an emergency with a non-fasted patient, the decision to proceed should be based on the urgency of the procedure and the intended depth of sedation
    - urgency of proposed procedure
        - in life or limb threatening situations (e.g. arrhythmia with cardiovascular compromise or orthopaedic procedure for distal limb ischaemia), the patient is unable to wait
        - the main question becomes the choice of sedation/anaesthetic technique
    - proposed depth and duration of sedation
        - longer periods of sedation, greater sedation depth and airway interventions may stimulate airway reflexes (coughing, hiccups or laryngospasm) and gastrointestinal motor responses (gagging or recurrent swallowing) leading to gastric distension, regurgitation or vomiting
    - patient factors
        - risk factors for delayed gastric emptying include:
            - raised intracranial pressure
            - hiatus hernia
            - gastrointestinal obstruction
            - previous upper GI surgery
            - recently injured people
            - patients receiving opioids
            - pregnancy
            - morbid obesity (higher intra-abdominal pressure and more common hiatus hernia)
    - for unstarved patients requiring deeper levels of sedation (e.g. for prosthetic hip relocation), maximise pre-oxygenation and consider employing apnoeic oxygenation (high flow oxygen via nasal cannulae) to minimise risk of bag valve ventilation which can insufflate the stomach and increase likelihood of regurgitation

        - the American College of Emergency Physicians recommends that procedural sedation should not be delayed based on fasting time
        - for sedating non-fasted patients, a senior Emergency Physician with level 2 sedation training should be present
    - high ASA grade patients
        - it may be appropriate to consult senior anaesthetic colleagues for patients ASA III or over
- pharmacological agents commonly used in the UK
    - morphine
        - 0.1mg/kg IV titrated to effect
        - onset 1-2 minutes
        - peak effect 10-15 minutes
        - duration 2-4 hours
    - fentanyl
        - 1-2mcg/kg IV titrated to effect
        - onset 1-2 minutes
        - peak effect 2-5 minutes
        - duration 20-30 minutes
    - nitrous oxide
        - up to 70%
        - onset 1-2 minutes
        - peak effect 2 minutes
        - duration: rapidly wears off
        - usually administered from premixed cylinders with oxygen at a concentration of 50% via a demand valve
        - if delivered by another method (e.g. anaesthetic machine) the concentration of oxygen must never be less than 30%
        - contraindicated in suspicion of pneumothorax, bowel obstruction, ruptured viscera or decompression illness due to volume expansion and pressure effects as it rapidly diffuses into closed air spaces
    - ketamine
        - 1mg/kg IV titrated to effect given over 60 seconds
            - 2-4mg/kg IM
            - reduced dose in the elderly (10-30mg IV)
        - onset 1-2 minutes
            - 2-5 minutes IM
        - peak effect 2 minutes
            - 5 minutes IM
        - duration 30 minutes
            - 90 minutes IM
        - dissociative anaesthetic and analgesia producing a trance-like state due to dissociation between the limbic and cortical systems
        - awareness of external stimuli is blocked, but the patient appears to be awake and have little cortical depression
        - relatively contraindicated in patients with airway instability or tracheal pathology, high predisposition to laryngospasm or apnoea, severe cardiovascular disease, CSF obstructive states, previous psychotic illness,

hyperthyroidism or thyroid medication use, globe injury or glaucoma, porphyria
    - midazolam
        - 0.02-0.1 mg/kg IV (adults), 0.025-0.05mg/kg IV (children) – titrated to effect
            - or 2-2.5mg with further 1mg doses after 2-5 minutes
            - total dose of more than 5mg not usually required in a healthy young adult, less than 3.5mg in adults over 60 and the chronically ill or debilitated
        - onset 1-2 minutes
        - peak effect 3-4 minutes
        - duration 30 minutes
        - fast acting water soluble benzodiazepine
        - no analgesic properties
        - should be prepared as a 1mg/ml solution
    - propofol
        - 0.5-1.0mg/kg bolus
            - further doses of 0.25 – 0.5mg/kg every 3-5 minutes
            - use 10-20mg boluses slowly in the elderly or chronically ill and debilitated
        - onset 1 minute
        - peak effect 1-2 minutes
        - duration 5-10 minutes
        - lipophilic agent thought to enhance GABA inhibitory neurotransmission
        - associated with profound hypotension and respiratory depression and frequent induction of deep sedation or general anaesthesia
- adverse events
    - propofol
        - respiratory depression
        - hypotension
        - decreased cardiac output
    - etomidate
        - nausea
        - vomiting
        - myoclonus
        - adrenal suppression
    - ketamine
        - tachycardia
        - hypertension
        - increased cardiac output
        - hypersalivation
        - emergence phenomenon
        - laryngospasm
    - midazolam
        - hypoventilation
        - hypoxaemia
        - hypotension
        - paradoxical stimulatory effect
    - fentanyl

        - pruritus
        - nausea
        - vomiting
        - respiratory depression
- using pharmacological agents
    - should be given IV if possible – allows quick and reliable onset and easier titration
        - IM route should be reserved for children and for adults with learning difficulties or behavioural problems
    - smaller doses should be used in the elderly, with extra time left for the drug to take effect (slower arm-brain circulation time)
    - patients with regular alcohol consumption may require larger doses and the therapeutic window may be narrow
    - all of the drugs can produce deeper sedation and even anaesthesia and combinations should be used with caution
        - opioids should be given first and adequate time left for them to take full effect before adding a sedative
- reversal agents
    - naloxone
        - titrate 100-200mcg every 1-2 minutes to reverse respiratory depression following opioid administration
        - extended monitoring required as the effects of naloxone may be shorter acting than the opioid
        - may precipitate withdrawal in opioid dependent patients
        - will antagonise analgesia in excess
    - flumazenil
        - can be given in small increments of 100-200mcg every minute to reverse respiratory depression following benzodiazepine use
        - use with caution in those on long-term benzodiazepines to avoid withdrawal symptoms
- safe practice
    - requirements for ED sedation
        - minimal sedation (Entonox)
            - one physician or ENP
            - current ILS or ALS or local equivalent
            - anywhere in the ED
            - pulse oximetry for monitoring
        - moderate sedation/analgesia (conscious sedation using IV agents)
            - one physician as sedationist AND one physician or ENP as operator AND one nurse
            - current ILS or ALS; local sign off for Level 1 sedation
            - resuscitation room
            - ECG, NIBP, pulse oximetry (capnography also recommended)
        - deep sedation/analgesia
            - one physician as sedationist AND one physician or ENP as operator AND one nurse
            - Royal College of Anaesthetists initial assessment of competence (IAC); local sign off for Level 2 sedation

                - resuscitation room
                - standards conforming to AAGBI guidelines for general anaesthesia (capnography mandatory)
            - dissociative sedation using ketamine
                - one physician as sedationist AND one physician or ENP as operator AND one nurse
                - Royal College of Anaesthetists initial assessment of competence (IAC); local sign off for Level 2 sedation
                - resuscitation room
                - standards conforming to AAGBI guidelines for general anaesthesia (capnography mandatory)
- training requirements
    - Level 1 sedation training
        - ASA grading
        - pre-procedural assessment including prediction of difficulty in airway management
        - pre-procedural fasting and risk benefit assessment
        - consent and documentation
        - drug selection and preparation: benzodiazepine/opioid combinations, intervals between increments and reversal drugs
        - monitoring, complications (e.g. hypoxia, hypotension) and rescue strategies
        - governance and audit
    - Level 2 sedation training
        - as per level 1
        - drug selection with emphasis on potential alternative strategies and/or lighter sedation
        - safe use of propofol
        - safe use of ketamine
- resuscitation room facilities (RCoA/RCEM)
    - full resuscitation equipment for basic and advanced life support with equipment and drugs checked daily and after each use (with checks recorded)
    - difficult airway equipment
    - continuous high flow oxygen with appropriate devices for administration
    - high pressure suction with appropriate suction catheters
    - a trolley capable of being tipped head down
    - monitoring: pulse oximeter, ECG, NIBP and continuous quantitative capnography
        - no guidance on how often to record observations, 5 minute intervals recommended
    - appropriate range of intravenous cannulae
    - an appropriate range of intravenous fluids and infusion devices
    - manual handling devices
    - time out checklist
        - including right patient, right side, monitoring, equipment, personnel, plan A, plan B etc.
- consent
    - a patient information leaflet should be available

    - informed consent should be taken where appropriate, including risks and benefits, side effects and alternative options
    - written consent is not statutory, but the consent process should be documented
- guidelines for discharge post-sedation
    - vital signs stable and within acceptable limits
    - sufficient time elapsed (up to 2 hours) after the last administration of reversal agents (naloxone, flumazenil) to ensure no risk of re-sedation
    - discharge into the presence of a responsible adult who will accompany them home and be able to report any post-procedure complications
    - patients and their escorts should be provided with written instructions regarding post-procedural diet, medications, driving, other activities such as operating machinery and signing legal documents, and a phone number to be called in case of emergency
- tips
    - use a pillow pre-emptively to achieve 'sniffing the morning air' position
    - tilt the head of the trolley upwards if an obsess patient is hypoventilating or bag mask ventilation required – potentially could be in this position from the outset
    - consider a 500ml fluid bolus pre-procedure in elderly patients on anti-hypertensive medication with a further bolus primed in anticipation of drop in blood pressure
- paediatric sedation
    - considerations
        - distraction techniques should be considered
            - e.g. music, hypnosis, confusing tactile stimuli, blowing away pain
            - use of films on a tablet computer
            - relaying information about the procedure in the form of a story
            - assistance of experienced nurse and capable parent for distraction
            - use of intranasal diamorphine as well as paracetamol and ibuprofen
            - inject warmed local anaesthetic slowly with a fine gauge needle
    - contraindications to ketamine procedural sedation
        - age less than 12 months
        - active upper or lower respiratory tract infection
        - active asthma
        - unstable or abnormal airway
        - tracheal surgery or stenosis
        - proposed procedure within the mouth or pharynx
        - patients with severe psychological problems such as cognitive or motor delay or severe behavioural problems
        - significant cardiac disease
        - recent significant head injury or reduced level of consciousness
        - intracranial hypertension with CSF obstruction
        - intra-ocular pathology
        - previous psychotic illness
        - uncontrolled epilepsy
        - hyperthyroidism or thyroid medication
        - porphyria
        - prior adverse reaction to ketamine
    - RCEM algorithm for ketamine sedation

        - discuss the benefits, risks and description of the procedure with the parent/guardian
        - obtain written consent
        - apply topical anaesthesia if time permits
        - employ distraction techniques
        - 1mg/kg IV slowly (over at least one minute)
            - alternatively 2.5mg/kg IM to the lateral aspect of the thigh
        - expect clinical effect in 1-2 minutes (IV) or 5 minutes (IM) – typically nystagmus and loss of response to verbal stimuli
        - supplemental 0.5mg/kg IV bolus PRN (or 1mg/kg IM)
        - allow the child to recover in a quiet observed area
    - potential complications of ketamine sedation
        - airway
            - noisy breathing
                - usually due to airway malposition
                - incidence of <1%
                - can normally be corrected by paying careful attention to optimal airway positioning
            - mild laryngospasm
                - occurs in 0.3%
                - reported incidence of intubation for laryngospasm is 0.02%
        - vomiting
            - incidence 5-10%
            - usually occurs during the recovery phase
        - lacrimation and salivation
            - 10%
        - transient rash
            - 10%
        - transient clonic movements
            - <5%
        - significant agitation
            - 1.5%
        - emergence phenomena
            - uncommon
            - not prevented with prophylactic benzodiazepines
            - benzodiazepines are recommended in the rare event of significant recovery agitation
    - management of adverse effects
        - vomiting
            - treat symptomatically
            - prophylactic ondansetron has a NNT of 9
            - 0.1mg/kg (max 4mg)
        - airway patency problems
            - apply simple airway manoeuvres
        - laryngospasm
            - if there is stridor, attempt airway repositioning

                - try gentle suctioning of any secretions and apply high flow oxygen with reservoir bag
                - if saturating appropriately, continue procedure
                - if stridor worsening or the child is desaturating, let them breathe via a bag valve mask and stop the procedure
                - if desaturating <92% start gentle BVM ventilation
                - if stridor becomes worse or complete airway obstruction develops, give suxamethonium 1mg/kg IV
                    - followed by tracheal intubation and urgent senior anaesthetic assistance
                    - continue gentle ventilation
    - training requirements and personnel
        - ketamine should only be used by clinicians experienced in its use and capable of managing any complication, particularly airway obstruction, apnoea and laryngospasm
        - children aged 12-24 months should only receive ketamine sedation from expert staff (usually a consultant)
        - at least three staff required:
            - doctor to manage the sedation and airway
            - clinician to perform the procedure
            - experienced nurse to monitor and support the patient, family and clinical staff
        - observations should be taken regularly and recorded
    - environment, equipment and recovery
        - in mixed departments, finding a quiet child-friendly area with appropriate paediatric life support equipment can be challenging
        - a quiet recovery area can also be difficult to find but is considered essential
        - monitoring includes ECG, BP, respiration and pulse oximetry
        - supplemental oxygen should be given and suction available
        - recovery should be complete between 60 and 120 minutes depending on dose and route used
        - child can be discharged when able to ambulate and vocalise/converse at pre-sedation levels
        - an advice sheet should be given to the parent/guardian advising rest, quiet, and supervised activity for the remainder of the day
            - the child should not eat or drink for two hours after discharge because of the risk of nausea and vomiting

PALLIATIVE AND END OF LIFE CARE

PalP1 advanced malignancy and end stage chronic disease

- background
    - end of life care is defined as care provided in the last year of life, although it may be for longer or shorter periods

    - end of life care may be delivered by disease-specific specialists and their teams, generalists such as primary care teams or hospital based generalists or by palliative care specialists in hospices, hospitals and community settings
    - supportive care is care that is given alongside and to enhance disease-modifying or life-prolonging therapies
    - palliative care is primarily conservative and aimed at giving comfort and maintaining quality of life in the last months of life
        - it aims to provide relief from pain and other distressing symptoms, integrate the psychological, social and spiritual aspects of a patient's care and continue to offer a support system to help people live as actively as possible until their death
    - patients to be considered for end of life care include people with:
        - advanced, progressive, incurable conditions
        - general frailty and co-existing conditions that mean they are at increased risk of dying within the next 12 months
        - existing conditions if they are at risk of dying from a sudden acute crisis in their condition
        - life-threatening acute conditions caused by sudden catastrophic events
- identifying adults approaching the end of life
    - systems should be used such as the Gold Standards Framework, the Amber Care Bundle or the Support and Palliative Care Indicators Tool (SPICT)
    - identification allows a holistic needs assessment to be carried out
    - support for carers can also be provided
- other aspects include:
    - providing the information the patient wants in an acceptable format
    - reviewing current treatment and optimising medication
        - this can also include reducing the number of unnecessary routine appointments, organising appointments near the patient's home, starting or stopping treatments
    - advance care planning should be offered
        - patients should have a copy of their plan that can go with them if they are admitted to hospital or a hospice
        - discussion might include:
            - the individual's concerns and wishes
            - their important values or personal goals for care
            - their understanding about their illness and prognosis
            - their preferences and wishes for types of care or treatment that may be beneficial in the future and the availability of these
    - information should be shared between services, using an electronic record if possible
    - health and social care practitioners should have skills to support in the following areas:
        - disease-specific
            - including symptom management, hydration and nutrition, access to medication
        - physical
        - psychological

        - social
            - including support and advice (e.g. signposting advice on benefits, finance and third-sector, local or national support services)
        - support with activities of daily living
            - including access to equipment and rehabilitation services
        - pastoral, religious and spiritual
        - cultural
    - transferring people between care settings
        - should be done as smoothly as possible, including transfer of equipment
    - out of hours care
        - patients, carers and other people important to them should have access to:
            - a healthcare professional available 24 hours a day, 7 day a week who can access the person's records and advance care plan and make informed decisions about changes to care
            - an out of hours end of life care advice line
            - an out of hours pharmacy service that has access to medicines for symptom management

PalC1 advanced care planning

- definition
    - 'Advance care planning is a process that supports adults at any age or stage of health in understanding and sharing their personal values, life goals and preferences regarding future medical care. The goal of advance care planning is to help ensure that people receive medical care that is consistent with their values, goals and preferences during serious and chronic illness.'
- terminology
    - advance statements
        - statements about the person's wishes with regard to medical treatment or social care in the future
        - may be verbal or written but should be documented where possible
        - not legally binding but should be taken into account where capacity is lost
        - may consist of specific wishes or more general statements about beliefs and values
    - advance decision to refuse treatment
        - legally binding statements regarding refusal of specific medical treatments in the event of a lack of capacity
        - may not relate to basic care such as provision of oral food and drink or prevention of bed sores
        - a person must be over 18 to make a valid advance decision to refuse treatment and must have the capacity to make the decision
    - lasting power of attorney
        - the nomination of another person to make decisions on one's behalf in the event of a loss of capacity
        - two types:
            - property and affairs LPA
            - health and welfare LPA

        - must be registered, at a cost, with the Office of the Public Guardian in order to be used
        - each must be signed by the applicant, a witness, the nominated attorney(s) and a signatory who confirms capacity
            - the latter can be a health professional but can also be anybody who has known the applicant for at least two years and does not benefit from the LPA
        - there must be a 'named person' who must be notified should the LPA be registered
        - a person must be over 18 and have the capacity to make the decision when applying for an LPA
        - Scotland and Northern Ireland have slightly different situations but they are similar
- advantages of advance care planning
    - allows the individual to think about what they would like to happen to them in the event of them losing capacity
    - may include areas such as:
        - use of IV fluids and parenteral nutrition
        - use of cardiopulmonary resuscitation
        - use of life-saving treatment (whether existing or yet to be developed) in specific illnesses where capacity might be impaired – e.g. brain damage, such as from stroke, head injury or dementia
        - specific procedures such as blood transfusion for a Jehovah's Witness
    - the topic may encourage the discussion of future options even if legally binding forms are not completed, for example with the patient's doctor, family and friends
        - it may help families prepare and reduce future conflicts
    - advance decisions are legally binding – if a doctor gives the patient life-saving treatment against their wishes expressed in an advance directive, they will face legal action
        - the advance decision must be valid (made when the patient had capacity) and applicable to current circumstances
- limitations of advance care planning
    - both doctors and patients may be unwilling to initiate discussions as they assume deterioration of their mental state in the future
    - there is debate about whether decisions made when healthy and with full mental capacity can apply to future situations of which the individual has no experience
    - an advance decision to refuse treatment cannot be used to:
        - ask for specific medical treatment
        - refuse basic care
        - request something that is illegal (e.g. assisted suicide)
        - choose someone to make decisions for you, unless that person is given LPA
        - refuse treatment for a mental health condition
            - doctors are empowered to treat under part 4 of the Mental Health Act
    - a doctor may decide not to follow an advance decision to refuse treatment if:
        - it involves life-saving treatment and has not been signed and witnessed
        - there is ambiguity in the wording of the directive
        - it is not applicable to the circumstances

        - it is felt to be invalid, such as:
            - it is not signed
            - there is reason to doubt authenticity (for example it was not witnessed)
            - it is felt that there is duress
            - there is doubt as to the person's state of mind at the time of signing
- initiating the discussion
    - ideally done by the patient's GP
    - triggers for discussion include:
        - the topic being brought up by the patient
        - diagnosis of a life-limiting disease
        - diagnosis of a condition likely to lead to impairment of capacity – e.g. mild cognitive impairment, dementia, motor neurone disease
        - deterioration in the condition of existing diseases – e.g. recurrence of a cancer or development of metastases
        - change in personal circumstances – e.g. move to care home, loss of partner
        - at agreed intervals where there is an existing plan
- implications of advance decisions for health professionals
    - basic principles:
        - the advance decision must be valid and applicable to current circumstances
            - if it is, health professionals must legally comply
        - in the event of the advance decision relating to refusing life-sustaining treatment it must be:
            - in writing (as opposed to verbal)
            - signed and witnessed
            - clearly stated that it applies even if life is at risk
        - what constitutes life-sustaining treatment is complex and ideally the advance decision should be very specific and clear about what is being refused
            - e.g. on some occasions antibiotic treatment may be considered life-saving, on others it may be to improve comfort
        - the health professional should try to establish:
            - if the decision has been withdrawn
            - if anything has occurred which might indicate the individual has changed or might change the decision
                - includes a change of circumstances which could not have been anticipated at the time of making the decision (e.g. new treatment or change in diagnosis/prognosis)
            - if the person has subsequently conferred the right to make the decision to another person by LPA
        - advance decisions to refuse treatment for mental disorder may not apply if the person is being detained (or likely to be detained) under the Mental Health Act
        - if the person has capacity to make the decision, the advance decision to refuse treatment is not valid and they should be consulted about their current wishes

        - people can withdraw or change their advance decisions at any time if they have capacity to do so
            - original decisions should be destroyed or the changes clearly documented
        - if an advance decision to refuse treatment is considered invalid, the person's wishes and best interests must still be paramount
        - health professionals are protected from liability if they do not follow an advance decision if they were unaware that it existed, or if they are not satisfied that one exists which is valid and/or applicable

PalC2 anticipatory medications

- background
    - anticipatory prescribing should be done with an individualised approach
    - prescribing needs should be discussed with the patient, those important to them and the multiprofessional team
    - medications should be reviewed as needs change
    - control of symptoms is one of seven key tasks from the Gold Standards Framework
        - the others are communication, co-ordination, continuity, continued learning, carer support and care of the dying
- when deciding which anticipatory medications to offer, things to take into account include:
    - the likelihood of specific symptoms occurring
    - the benefits and harms of prescribing or administering medications
    - the benefits and harms of not prescribing or administering medications
    - the possible risk of the person suddenly deteriorating (for example catastrophic haemorrhage or seizures) for which urgent symptom control may be needed
    - the place of care and the time it would take to obtain medications

PalC3 end stage organ failure

- heart failure
    - background
        - around 40% of patients die within a year of diagnosis
        - quality of life may be poor with a heavy burden of symptoms and many psychological and social needs
    - place of care
        - most appropriate place does not depend on the aetiology of the terminal condition
        - many/most people would prefer to die at home
        - nurse-led community management can provide support with:
            - regular follow-up and assessment including bloods to detect early clinical deterioration
            - continued adjustment and optimisation of therapy according to agreed protocols
            - promotion and support of self-management, including daily monitoring of weight
            - education covering both pharmacological and non-pharmacological aspects of care, including exercise and nutrition advice

                - acting as an intermediary between the patient and other professionals – e.g. cardiologists and the primary healthcare team
                - provision of support for patients and their carers
        - patient needs
            - symptoms of CHF include:
                - fatigue and breathlessness
                    - most common limiting and distressing complaints
                    - position of patient (supine/prone) should be considered
                    - oxygen may be helpful
                    - morphine is often effective if dyspnoea remains a problem
                        - helps to relieve distress and can improve left ventricular failure, does not cause undue suppression of the respiratory drive
                - depression
                    - may occur in a third of patients
                    - often overlooked
                    - should be sought and treated if found
                    - tricyclic antidepressants should be avoided in heart failure
                - pain
                    - common, especially in the terminal stages
                    - once common causes is stretching of the capsule of the liver
                    - should be managed in the usual way for palliative care except that NSAIDs should be avoided
                    - morphine or diamorphine can be of value for both pain and dyspnoea
                - nausea and decreased appetite
                    - common, may result in nutritional deficit
                    - factors include decreased hunger sensations, diet restrictions, fatigue, shortness of breath, nausea, anxiety, sadness
                    - in the elderly, early satiety, decreased taste and smell and eating alone may contribute
        - management
            - communication
                - including talking about prognosis at a time when the patient is able to process the information
            - drug management
                - use of maximum tolerated doses to control heart failure
            - nutrition
                - small, frequent, easily digested and appetising meals
- end stage renal disease
    - background
        - defined as patients whose eGFR is <15ml/min or who are requiring dialysis to avoid life-threatening complications
    - peritoneal dialysis
        - questions to consider
            - type

        - automated peritoneal dialysis (APD)
            - fluid is repeatedly cycled in/out of the abdomen
        - continuous peritoneal dialysis (CPD)
            - around 4-5 exchanges are performed over the course of a day
            - the patient uses gravity to drain the fluid in/out
    - timing
        - more recently sited tubes are more likely to result in peritonitis
    - strength and volume of dialysate bags
        - gives an indication to the renal team of how much fluid is drawn off and the degree to which electrolytes, uraemia and acidosis are altered
    - person doing the exchanges
        - patient, relative or carer
    - problems with the hygiene regimen
        - whether anyone has forgotten to wash their hands or clean the exit site
        - any obvious contamination episode
    - previous catheter exit site infections or peritonitis
    - recent GI or biliary tract infection
        - increases risk of peritonitis
    - constipation
        - can cause the catheter to move or not work properly
        - can cause bacteria to translocate across the bowel wall
        - both risk factors for CAPD peritonitis
    - cleanliness of exit site
    - tenderness of the tunnel
- PD peritonitis
    - abdominal pain is the most common presentation (but 1 in 20 do not have abdominal pain)
    - fever is only present in a third
    - the dialysate fluid almost always becomes cloudy but this may lag behind other symptoms
    - blood cultures are often negative unless the patient is systemically unwell
        - a sample of dialysis fluid must be sent for microscopy, cell counts and culture
        - the PD catheter should only be accessed by trained individuals, and renal staff where available
    - management
        - intraperitoneal antibiotics are first line
            - these should be administered by the renal team, who should be involved early
            - a typical regime is intraperitoneal vancomycin and gentamicin and/or oral ciprofloxacin
    - pitfalls include:

        - overlooking a surgical cause for abdominal pain
            - low threshold for involving the surgeons
        - do not attempt to drain peritoneal fluid – this should be done by a specialist
        - if the patient has features of life-threatening sepsis, they will also require IV antibiotics and the catheter may need to be removed
- access types
    - fistula
        - access of choice in a patient needing long-term haemodialysis
        - surgically created by anastomosing an adjoining artery and vein
        - common fistulae are radiocephalic, brachiocephalic and brachiobasilic
        - takes several months to mature into a useable high pressure system
        - can be recognised by its tortuous and bulging appearance
        - usually very superficial and feel squishy
        - there should be a palpable thrill if working properly
        - there will be an associated scar if you look carefully
        - they can last for many years and have a low infection rate
    - graft
        - an artificial conduit between a vein and an artery
        - usually used if the patient does not have adequate vessels to create a fistula
        - feel like a tube
        - often straight, although can be looped and are deeper under the skin than a fistula
        - there are two small associated scars
        - they can normally be used a few weeks after insertion
        - they do not normally last as long as a fistula and have a higher infection rate
        - a functioning graft will also have a thrill
    - tunnelled lines
        - usually a temporary bridge whilst more permanent access is maturing
        - can be used immediately following insertion
        - the right internal jugular is the preferred site
        - subclavian lines have a higher chance of clotting off or producing stenoses
        - femoral lines are a high infection risk
        - occasionally patients will have ‘permanent’ lines as their source of access if all other options have been exhausted
        - tunnelled lines have the highest risk of clotting and infection
- bleeding fistula
    - preparation
        - resuscitation room if possible
        - have enough pairs of hands – at least a nurse and a second pair of hands

- PPE – be prepared for mess (eye and face protection, aprons, gloves)
- consider giving the renal and vascular team advance warning

- management:
  - firm pressure with one finger on the bleeding site, over the exact point where the fistula is spurting
  - a temporising measure (especially if short of hands) is to place a gallipot over the fistula and secure with tape
  - maintain firm pressure for 5-10 minutes without releasing pressure or sneaking a look
  - re-examine the fistula after 10 minutes of pressure
    - the technique will work in the majority of cases
    - the patient should be observed for 1-2 hours
      - consider admission if evening/night time, the patient will be at home alone or is frail
  - manage as catastrophic haemorrhage if required
  - if bleeding continues:
    - ensure someone keeps single finger pressure in place
    - if recently dialysed, consider applying protamine soaked gauze
    - consider using a haemostatic aid such as Gelofoam if available
    - if the patient has any other bleeding tendency (including anticoagulants and antiplatelets) – speak to the haematologists early
      - all renal patients are anticoagulated during dialysis, have poorly functioning platelets and an elevated bleeding time secondary to uraemia so will bleed profusely
    - involve a vascular surgeon early – the fistula may need to be sutured if usual measures fail
    - applying a tourniquet is an absolute last resort
      - it will work but almost certainly result in thrombosis and fistula failure
- golden rules when dealing with grafts and fistulas
  - try not to touch a renal patient's access site unless they are in extremis
    - do not take blood from them and do not administer drugs or fluids via them
  - do not use a blood pressure cuff or tourniquet on the same arm as the graft/fistula – risk of clotting the access
  - the access is the patient's lifeline and losing it is a disaster
- if a patient is in extremis and using the fistula for circulatory access is being considered:
  - is there really no alternative? (e.g. US-guided peripheral access or IO)
  - do not use a tourniquet

- clean the site very well
- avoid puncturing the back wall of the vessel
- secure the IV catheter very carefully
- remember that this is a high pressure system and a pressure bag will be required to get fluid into the patient
- document the presence of a thrill before and after the procedure
- 10-15 minutes of firm pressure will be required on removal of the catheter

▪ fistula infection
- renal patients are relatively immunocompromised and the access site is frequently accessed so there is a high risk of infection
- there may or may not be redness around the access site
- there may or may not be pus
- there needs to be a very high index of suspicion
- line or access site infections can seed and cause more unusual infections such as endocarditis, osteomyelitis and discitis

▪ fistula thrombosis
- there is loss of thrill or bruit from the graft or fistula
- clinical diagnosis but can be confirmed with US doppler
- feel the fistula/graft, then listen with a stethoscope or hand-held doppler
    - continuous flow should be heard
- a high pitched bruit may indicate imminent stenosis
- more likely to occur in a graft than a fistula
- there is usually 24-48 hours to declot the access site, often done by interventional radiologists
- from an ED perspective, early diagnosis and referral is important
- in case of access failure, it is important to check U&Es so the renal team know when dialysis is required

o haemodialysis

▪ history
- what sort of access do they have (fistula/graft/tunnelled line)
    - have they had problems with dialysis access in the past
- how frequently and for how many hours they dialyse for
    - most people, unless on home dialysis, dialyse three times a week on set days
- when they last dialysed
    - helps predict problems such as fluid overload and hyperkalaemia
- detailed fluid balance history
    - what their dry weight is (clinical estimation of their weight when euvolaemic)
    - what their fluid restriction is
    - how much fluid is normally removed at dialysis
    - whether they still make any urine
- how dialysis has been going recently
    - whether there have been any technical problems

        - whether they have felt unwell whilst on dialysis
    - whether they are on the transplant list
- hypotensive episodes during dialysis
    - differential
        - fluid shifts
            - removal of too much fluid or fluid removed too quickly
        - sepsis
        - bleeding
            - external (e.g. bleeding access site)
            - internal (head, gut, pericardium)
        - cardiac tamponade
            - due to bleeding or uraemia
        - arrhythmia
            - e.g. due to potassium shifts
        - hypersensitivity reaction
            - allergic or anaphylactic
        - acute coronary syndrome
        - air embolism
        - dialysis hypotensive syndrome
    - history
        - whether the patient was unwell beforehand
            - e.g. symptoms of infection or tamponade
        - at what point during dialysis the patient became hypotensive
            - at the beginning of dialysis hyperkalaemia is more likely
            - at the end of dialysis, hypotension due to fluid shifts is more likely
        - whether the patient has any symptoms of infection
            - fever, malaise, systemic upset, focal symptoms of infection
        - any evidence of bleeding
            - obvious bleeding site, signs of shock
        - any symptoms or signs of allergic reaction
            - rash, wheeze, breathlessness
        - symptoms or signs of air embolism
            - e.g. chest pain, breathlessness, hypoxia, tachycardia if the embolus travels to the heart; syncope or seizures if the embolus travels to the brain
        - symptoms indicative of a cardiac cause
            - chest pain, palpitations, breathlessness
    - management if infection suspected
        - things that should be done:
            - search for a source of infection
            - culture the patient
            - administer broad spectrum antibiotics as soon as possible

    - things that probably should not be done
        - immediate removal of the tunnelled line
        - catheterisation
        - stat 1 litre fluid bolus
        - central line insertion
    - sepsis is the second most common cause of death in end stage renal patients (cardiac causes being the commonest)
    - there is little point in catheterising as they will not produce urine
    - lactate may have been removed if recently dialysed
    - cautious 250ml fluid boluses may be given
    - antibiotics should be dose adjusted for renal function
        - if patients still pass urine in less critical circumstances and have some residual renal function, nephrotoxic medications should still be avoided
            - any residual renal function will be very important to the patient and may mean slightly less severe daily fluid restriction
        - if the patient is very unwell, a risk/benefit assessment for nephrotoxic antibiotics needs to be carried out
    - if a line or graft infection is suspected, every effort should be made to preserve the access site
        - any access removal would be by joint decision from renal and critical care teams
- bleeding
    - renal patients have a tendency to bleed, including intracranial or intra-abdominal bleeds
    - they also have an increased tendency for thromboembolism
- cardiac tamponade
    - secondary to bleeding or uraemia
    - early ultrasound should be considered unless there is an obvious other cause
    - treatment in the compromised patient involves optimising right ventricular filling with fluids and pericardiocentesis
    - emergency dialysis is the treatment of choice in uraemic tamponade
- arrhythmia
    - hyperkalaemia may cause arrhythmias or activate an ICCD if the patient has one (ECG changes misinterpreted as VT)
    - early ECG and knowledge of the usual potassium level is useful
- hypersensitivity reaction
    - may be against components of the dialysis membrane or to foreign substances in the extra-corporeal circuit
    - standard allergy/anaphylaxis management

- acute coronary syndrome
    - dramatic fluid shifts place stress on the heart and can precipitate symptoms of cardiac ischaemia
    - 50% of all deaths in end stage renal disease are cardiac in origin
- air embolism
    - occurs when air is inadvertently let into the system during dialysis
    - all symptoms may be explained by alternative causes so high index of suspicion required
    - the biggest clue is the temporal relation to dialysis
    - management is uncertain but includes:
        - left lateral position to try and prevent further propagation of the air through the right side of the heart and into the pulmonary arteries
        - try and suck any remaining air out of the catheter (hopefully already done by the dialysis team)
        - if in arrest, chest compression may break up the embolus, and a prolonged period of resuscitation should be considered
        - in extremis, a thoracotomy could be considered to try and suck air out of the right ventricle
- dialysis hypotension syndrome
    - multifactorial and poorly understood
    - not an ED concern

- end stage COPD
    - long term oxygen therapy
        - likely to be lifelong once started
        - usually given for 15 hours a days, including overnight when arterial hypoxaemia worsens during sleep
            - there is no benefit if used for <15 hours
        - candidates include people with:
            - very severe airflow obstruction (FEV_1 <30% predicted)
            - cyanosis
            - polycythaemia
            - peripheral oedema
            - raised jugular venous pressure
            - oxygen saturation 92% or less on air
        - assessment will include two ABGs at least 3 weeks apart
            - LTOT should be offered if PaO_2 <7.3kPa when stable (or <8kPa if they have peripheral oedema, polycythaemia or pulmonary hypertension
        - inappropriate therapy may cause respiratory depression
        - risk of falling over equipment may outweigh the benefit of LTOT
        - long term NIV is an option for patients who are acidotic and hypercapnic on LTOT
    - ambulatory oxygen therapy

- may be offered to people on LTOT who wish to use oxygen outside the home
- BTS recommends ambulatory oxygen therapy:
    - should not be routinely offered to patients not eligible for LTOT
    - should not be routinely offered to patients on LTOT
    - should only be offered to patients on LTOT if they are mobile outdoors
    - should be offered for use during an exercise or pulmonary rehabilitation programme

- short-burst oxygen therapy
    - used for relief of breathlessness not relieved by other treatments
    - used intermittently at home for short periods (10-20 minutes)
    - should not be used for patients with mild to moderate COPD
- patients on home oxygen therapy should be monitored with ABGs and given education and support
- travel
    - reduced pO_2 in airline cabins increases hypoxia in patients who have hypoxia at sea level
        - BTS: commercial air travel is contraindicated for patients with usual oxygen requirements >4L/minute at sea level
    - most major airlines can provide supplemental in-flight oxygen and assistance with embarkation if arranged in advance
    - LTOT can usually be arranged temporarily from a local chemist during a holiday but many patients can manage well without LTOT for several days

PalC4 pain management

- background
    - pain occurs in up to 70% of patients with advanced cancer and about 65% of patients dying of non-malignant disease
    - the patient should be the prime assessor of their pain
    - things that lower pain tolerance include:
        - discomfort
        - insomnia
        - fatigue
        - anxiety
        - fear
        - anger
        - boredom
        - sadness
        - depression
        - introversion
        - social abandonment
        - mental isolation
    - things that improve pain tolerance include:
        - relief of symptoms

        - sleep
        - rest or physiotherapy
        - relaxation therapy
        - explanation/support
        - understanding/empathy
        - diversion
        - listening
        - elevation of mood
        - finding meaning and significance
        - social inclusion
        - support to express emotions
- assessment
    - should include:
        - physical effects or manifestations
        - functional impact of pain
        - psychosocial factors
        - spiritual aspects
    - at least daily monitoring with visual analogue, numerical or verbal rating scales to allow treatment to be modified promptly where pain is inadequately controlled
        - self-assessment should be used wherever possible, even in patients with cognitive impairment
- management
    - over 80% of cancer pain can be treated with inexpensive oral drugs
    - WHO analgesic ladder
        - step 1 (pain <3/10)
            - paracetamol and NSAIDs
        - step 2 (pain 3-6/10)
            - weak opioid + paracetamol + NSAIDs +/- adjuvant analgesic
        - step 3 (pain >6/10)
            - strong opioid + paracetamol + NSAIDs +/- adjuvant analgesic
    - adjuvant analgesics for cancer pain:
        - NSAIDs
            - bone pain
            - soft tissue infiltration
            - hepatomegaly
        - corticosteroids
            - raised intracranial pressure
            - soft tissue infiltration
            - nerve compression
            - hepatomegaly
        - antidepressants and anticonvulsants
            - nerve compression or infiltration
            - paraneoplastic neuropathies
        - bisphosphonates
            - bone pain
        - ketamine
            - refractory pain

                - neuropathic pain
                - ischaemic limb pain
    - patients should be told that it is easier to prevent than to relieve pain and medication should be regular
    - breakthrough medication should also be prescribed
        - ideally to be taken before a potential pain-provoking activity
    - treatment should be as simple as possible with the minimum number of drugs in the most acceptable form and dose intervals
    - regular review is required
    - anticipatory prescribing should be done, with equipment and adequate quantities of drugs available
        - a 'just in case' box can contain analgesia, antiemetics and sedatives
    - nerve blocks or regional anaesthesia may be considered for localised pain
- analgesic types
    - non-opioids
        - paracetamol
            - weak analgesic
            - very few side effects
            - has a dose-sparing effect with codeine
        - NSAIDs
            - particularly useful for bone pain
            - main side effect is GI bleeding
                - PPI (standard dose), H2-receptor antagonist (double dose) or misoprostol can be co-prescribed
    - weak opioids
        - includes codeine phosphate, dihydrocodeine and tramadol
    - strong opioids
        - oral morphine
            - initially given as 4-hourly immediate release tablets or elixir or 12 hourly sustained release morphine, depending on patient preference
            - extra doses for breakthrough pain as required
            - a typical starting dose for an opioid-naïve patient is 4-hourly 5-10mg immediate release morphine or 12-hourly 10-15mg sustained release morphine
            - after 24 hours, the previous day's intake should be totalled and divided by 6 to provide 4 hourly doses or 2 to provide 12 hourly doses
        - maintenance dose
            - NICE recommends oral sustained-release morphine as first line
            - breakthrough pain should be treated with immediate-release morphine at 1/6 total daily dose
            - buccal or intranasal fentanyl may be considered for incident or episodic pain such as unusual activity or dressing changes due to swift absorption and rapid onset
- common problems of opiates
    - constipation

        - prophylactic laxative should be prescribed
            - there is no particular preference
            - a combination of drugs with different modes of action may be needed for resistant constipation
    - sedation
        - usually settles within a few days
    - nausea and vomiting
        - occurs in up to 40% of opioid-naïve patients
        - may settle within a few days
        - an antiemetic such as metoclopramide 10mg TDS or haloperidol 1.5mg ON should be used
    - dry mouth
        - good mouth care
            - frequent sips of iced drinks
            - dental floss
            - eating pineapple chunks
            - saliva replacements or stimulants
    - histamine release
        - pruritus
            - antihistamine
        - bronchoconstriction
            - IV/IM antihistamine and bronchodilator
            - switch to pharmacologically distinct opioid such as methadone
    - toxicity
        - e.g. agitation, hallucinations, confusion, vivid dreams, myoclonic jerks
        - may be caused by worsening renal or hepatic function
    - morphine intolerance
        - may be affected by:
            - responsiveness of the pain to opioids
            - previous exposure to opioids
            - rate of dose of titration
                - start low and titrate upwards
            - additional medication
            - concomitant disease
            - genetic factors
            - renal and hepatic function
    - alternatives include hydromorphone, methadone or oxycodone
    - in patients with poor or worsening renal function:
        - consider dose reduction and/or increased dose interval
        - change from sustained release to immediate release oral formulation
        - consider switch to alfentanil, buprenorphine or fentanyl
            - opioids of choice if eGFR <30
        - frequent monitoring and review
        - specialist input
    - parenteral routes
        - subcutaneous

- may be used first line if oral opioids not suitable and analgesic requirement unstable
- syringe drivers
    - injection site should be changed every 2-3 days
    - diamorphine is around 3 times as powerful as oral morphine
    - other drugs can be mixed with diamorphine in the syringe

- transdermal
    - for stable pain (dose cannot be changed quickly)
    - transdermal fentanyl is 100-150 times more potent than oral morphine
    - patches are worn for 72 hours
        - steady state is reached after a variable time
    - immediate release oral morphine should be continued for 12 hours after the first patch is applied
    - if analgesia lasts less than three days, the patch strength should be increased
    - elimination half life on removal of patch is almost 24 hours
    - transdermal buprenorphine is 75 times more potent than morphine
        - available as 4 and 7 day patches

- common problems
    - 10% of patients at the end of life have 'difficult pain'
        - pain poorly responsive to opioids
        - episodic and breakthrough pain despite background opioid analgesia
        - caused or aggravated by non-physical factors such as psychological or social distress
    - help can be sought from specialist palliative care teams, Macmillan teams and GPs with a special interest in palliative care

PalC5 physical symptoms other than pain

- dyspnoea
    - background
        - the distressing awareness of the process of breathing
            - either the frequency or the effort involved
        - frightening, and one of the most common symptoms in palliative care
        - present in 90-95% of patients with COPD, 60-88% of those with chronic heart failure, 10-70% of those with cancer and is also common in end-stage kidney disease and AIDS
    - aetiology
        - the drive to breathe and stimulus for dyspnoea is complex and multifactorial:
            - main driving force is pCO_2 rather than pO_2 (except in hypercapnic COPD), although significant hypoxia can augment the hypercapnic drive
            - mechanical stimuli such as pulmonary stretch and proprioceptive input from the chest wall and diaphragm are important
            - pyrexia stimulates the thalamus and can cause tachypnoea

- emotions (e.g. anxiety, fear, anger) and arousal state also modulate breathing

- causes of dyspnoea
  - potentially reversible or partially reversible with further treatment
    - infection
    - bronchoconstriction
    - pleural or pericardial effusion
    - pneumothorax
    - pulmonary embolism
    - cardiac failure/dysrhythmia/anaemia
    - panic or psychological disorder
    - superior vena cava obstruction
    - lymphangitis
    - ascites
  - irreversible
    - progression of disease (e.g. malignant infiltration, fibrosis, congestion) leading to diminished lung function
    - progression of neurological or muscular disease preventing adequate ventilation
- assessment
  - history
    - previous or pre-existing conditions
    - recent treatments (e.g. radiotherapy)
    - recent medication changes
    - breathlessness history:
      - rapidity of onset (insidious or sudden) and time course (acute, chronic, intermittent)
      - associated symptoms
        - e.g. cough, haemoptysis, stridor, wheeze, pain, fatigue
      - relationship to exercise
      - precipitating factors
        - e.g. posture, exertion, pollen, emotion
      - impact on simple daily activities
        - e.g. talking, washing, toileting, dressing, sleeping
      - role of carers
      - episodes of panic or anxiety
      - impact on quality of life or any mood disturbance
      - any known relieving factors
  - examination
    - effort and efficacy of breathing
      - e.g. depth of breathing, use of accessory muscles, expectoration of secretions
    - observe the patient at rest and, where appropriate, walking or undertaking a task
    - assessment of degree of anxiety
    - cardiac status

        - rhythm, rate, cardiac murmurs, signs of cardiac failure
      - clinical signs of infection, effusion, anaemia, cyanosis
      - examination of the respiratory system based on known disease, looking for signs of stridor and progressive disease
  - investigations
    - unnecessary investigations should be avoided
      - consider stage of disease, risk-benefit ratio and the wishes of the patient and their family
    - possible investigations include:
      - CXR
      - spirometry
      - ECG to exclude arrhythmia
      - FBC to exclude anaemia
      - pulse oximetry
- management
  - general
    - addressing breathlessness as soon as it becomes apparent rather than waiting until it is well established
    - explore ongoing fears (e.g. choking, suffocation, dying in sleep)
    - useful strategies:
      - upright positioning with support
      - keeping the room cool
      - moving air from a fan (hand-held or stationary) or open window to provide psychological relief
      - teaching and use of breathing exercises and relaxation methods
        - breathing retraining taught by physiotherapists or clinical nurse specialists
      - modification of lifestyle
        - reducing non-essential activities whilst trying to maintain mobility and independence as far as possible
      - encouraging exertion to the point of breathlessness to build tolerance and maintain fitness
      - dietary modifications with small frequent drinks and meals
        - dietician input may be helpful
      - attention to oral hygiene if mouth breathing or using supplementary oxygen
      - some patients may find complementary therapies such as aromatherapy, hypnosis and acupuncture helpful, but evidence base is weak
  - medical interventions
    - primary care
      - treatment of reversible or partly reversible causes e.g.:
        - optimising treatment of asthma, COPD, heart failure
        - treatment of infections which will aggravate dyspnoea

- secondary care
    - blood transfusion
        - anaemia can exacerbate dyspnoea on exertion
    - treatment of underlying disease
        - pleural effusion tapping +/- pleurodesis
        - ascites tapping
        - management of SVC obstruction with high dose steroids, radiotherapy, chemotherapy, dilatation, stenting or anticoagulants
        - management of tumours impinging on the trachea or bronchus – e.g. steroids, external radiotherapy, chemotherapy, endobronchial treatments such as laser, radiotherapy, stenting, cryotherapy, balloon dilatation
    - emergencies such as PE or pneumothorax may justify admission for active treatment

- drugs
    - opiates
        - can provide good symptom relief with negligeable respiratory depression
        - oral morphine
            - started at a low dose and titrated according to response and side effects
            - starting dose 5mg 4-hourly PRN, larger dose if already on morphine
        - continuous subcutaneous infusion when oral route no longer available – may be combined with a benzodiazepine
        - IV or SC diamorphine often used for more rapid relief in extreme dyspnoea and distress
        - anti-emetics and laxatives may be required
    - anxiolytics
        - no evidence for use of benzodiazepines for relief of dyspnoea associated with cancer or COPD
        - can be used as second or third line when opioids and non-pharmacological interventions have failed
        - options include sublingual lorazepam (0.5mg 4-6 hourly) or subcutaneous midazolam (5-20mg over 24 hours)
        - tricyclic antidepressants and SSRIs may be helpful, especially for panic attacks
    - oxygen therapy
        - unclear role and no convincing evidence of benefit for palliation of dyspnoea
        - should only be used where the patient is shown to be hypoxic
        - a trial of short burst oxygen therapy (e.g. 2L/minute for 15-30 minutes) may be considered if appropriate and no risks such as smokers in the house
    - respiratory secretions

        - first line treatment is hyoscine butylbromide 20mg SC hourly
        - other options are:
            - glycopyrronium bromide 200 microgram 6-8 hourly
            - hyoscine hydrobromide 400 microgram 2-hourly
- nausea and vomiting
    - background
        - most common in late stage AIDS patients (43%)
        - prevalence increases in last 1-2 weeks of life (up to 70%)
        - 15-30% of patients given morphine for chronic cancer pain have long-term nausea
    - causes
        - four main sites of activity
            - the vomiting centre
                - situated in the brainstem
                - has histamine (H1), acetylcholine (ACh) and 5-hydroxytryptamine (5-HT2) receptors
            - the chemoreceptor trigger zone
                - located in an area of the brain that has no blood-brain barrier
                - various drugs, toxins and metabolites can access the site
                - has dopamine (D2) and 5-HT3 receptors
            - the cerebral cortex
                - multiple receptors which can be triggered by anxiety
                - mechanoreceptors in the meninges sensitive to changes in intracranial pressure
            - the vestibular system
                - changes in movement or disease of the ear may stimulate the ACh or H1 receptors, triggering nausea or vomiting
            - gut and serosal surfaces in the viscera
                - 5-HT3 receptors in the gut are stimulated by drugs, radiotherapy and bacterial endotoxins
                - H1 and ACh receptors in the gut and serosal surfaces of other viscera are stimulated by mechanical distortion
    - underlying causes and mechanisms
        - irritation or stretching of the meninges
            - e.g. raised intracranial pressure caused by intracranial tumour
            - mechanisms not known, may involve meningeal mechanoreceptors
            - features:
                - headache and nausea on lying flat
                - focal neurological signs
                - papilloedema
            - investigations:
                - CT/MRI
        - pelvic or abdominal tumour
            - e.g. mesenteric metastases, liver metastases, ureteric obstruction, retroperitoneal cancer
            - caused by stretching of mechanoreceptors

- features:
    - poorly localised pain, with or without radiation, may be present
- investigations:
    - radiology

- bowel obstruction secondary to malignancy
    - e.g.
        - mechanical
            - intrinsic or extrinsic by tumour
        - functional
            - disorders of intestinal motility secondary to malignant involvement of nerves, bowel muscle or blood supply
        - paraneoplastic neuropathy
    - caused by stretching of mechanoreceptors
    - features:
        - usually insidious onset and partial obstruction
        - abdominal pain in 90%
            - superimposed colic in 70%
        - less distension in high obstruction or if bowel stuck down by omental metastases
        - vomiting, often copious, is an early feature in high obstruction and a later feature in large bowel obstruction
- gastric stasis
    - e.g. drugs (anticholinergics, opioids), mechanical obstruction to gastric emptying (tumour, gastritis, peptic ulcer, hepatomegaly), autonomic failure (e.g. advanced diabetes)
    - caused by gastric mechanoreceptors
    - features:
        - fullness
        - epigastric pain
        - acid reflux
        - hiccups
        - early satiety
        - large volume vomiting with little preceding nausea
        - symptoms relieved by vomiting
- chemical/metabolic
    - e.g. drugs (anti-epileptics, opioids, antibiotics, cytotoxics, digoxin), metabolic (hypercalcaemia), toxins (tumour necrosis, bacterial toxins)
    - caused by chemoreceptors in the trigger zone
    - features:
        - onset of symptoms may coincide with starting medication
        - drowsiness may be the only feature in 50% of those with hypercalcaemia
            - confusion is common
- anxiety-induced

                - concern about diagnosis, treatment, symptomatology, social issues, anticipatory emesis with cytotoxics
                - caused by multiple receptors in the cerebral cortex
                - usually a diagnosis of exclusion and suggested by the symptoms and signs of stress
            - movement related
                - e.g. abdominal tumours, opioids, disease affecting the vestibular system
                - caused by stretch of mechanoreceptors by tumours, increase in vestibular sensitivity by drugs, disturbance of vestibular function by disease
    - history
        - timing of symptoms
            - after meals
                - gastric stasis
            - on movement
                - e.g. vestibular disease
            - when lying flat
                - meningeal irritation or raised ICP
        - food and fluid intake
        - drugs, including OTC and alternative therapies
        - pain
        - bowel habit
        - urinary output
        - affect on daily life
    - examination
        - assessment of hydration
        - signs of infection, e.g. fever
        - jaundice
        - neurological examination including fundoscopy to assess for papilloedema
        - rectal examination
        - abdominal examination
            - tenderness
            - distension
            - ascites
            - masses
            - hepatomegaly
    - possible investigations
        - U&Es
        - calcium
        - LFTs
        - FBC and differential
        - urine culture
        - abdominal imaging (US/x-ray)
        - endoscopy
        - CT/MRI
    - management

- requires MDT approach
- simple advice
    - size and type of meal
    - advice on fluid intake
    - timing of medication
- attention to environment
    - bowls, tissues and drinking water within reach
- oral hygiene and mouthwashes for patients unable to drink
- consideration of syringe drivers or patches if nausea and vomiting are preventing other medications from being effective
- review of medication with stopping or changing of any contributing factors
- correction of reversible causes:
    - rehydration and bisphosphonates for hypercalcaemia
    - IV fluids may correct uraemia in some patients
    - PPIs or H2 receptor antagonists for gastric ulceration or gastritis
    - antibiotics for infection
    - laxatives or enemas for constipation
    - corticosteroids may reduce tumour size or surrounding oedema
    - anxiolytics may have a role in some patients
- specific situations
    - irritation or stretching of the meninges
        - consideration of radiotherapy if the patient has raised ICP
        - high dose dexamethasone (max 16mg/day for 4-5 days then reducing to 4-6mg/day)
        - cyclizine 25-50mg TDS or levomepromazine once at bedtime (2.5-5mg SC or 12.5mg PO)
    - pelvic or abdominal tumour
        - cyclizine
            - blocks ACh and histamine H1 receptors in the vomiting centre that are triggered by the mechanoreceptors in the abdominal and pelvic viscera
            - dexamethasone can be added if vomiting persists
    - malignant bowel obstruction
        - functional or partial obstruction
            - balancing of preservation of bowel motility with the prevention of colic
            - stop osmotic and stimulant laxatives
            - titration of docusate to produce a comfortable stool without colic
            - avoiding high fibre foods
            - food and fluids at regular intervals in small amounts
            - if the patient is passing flatus and does not have colic, a prokinetic anti-emetic such as metoclopramide or domperidone should be tried
                - block dopamine D2 receptor activity in the gut

- prokinetics should not be given with antimuscarinics (e.g. cyclizine, hyoscine) as they are competitively blocked by them
- if the patient develops colic, the prokinetic should be stopped and complete obstruction assumed

        - haloperidol is a specific dopamine D2 receptor antagonist that has a profoundly inhibitory effect on the CTZ
        - olanzapine may be useful but is not licensed yet
    - complete obstruction
        - cyclizine is the first line treatment – blocks the stimulation of the vomiting centre via the vagal afferents, which happens in complete obstruction
        - levomepromazine is second line
        - large volume vomiting should be treated with an antisecretory drug
        - NG tube if there is gastric outflow obstruction with rapid dehydration – can be removed as soon as there is control with medication
        - IV or SC rehydration
        - octreotide for complete distal obstruction
        - venting gastrostomy may be an option if there is ongoing need for an NG tube
        - stenting or corticosteroids may be considered if vomiting persists
- gastric stasis
    - prokinetics such as metoclopramide or domperidone are first line
    - therapies which reduce gastric secretions can be added, such as ranitidine or octreotide
- chemically/metabolically induced nausea
    - haloperidol for drug induced nausea, kidney disease and hypercalcaemia
    - prophylactic prokinetic when introducing/titrating morphine
    - ondansetron may sometimes be helpful
- anxiety-induced nausea
    - may be helped with communication, psychological and spiritual support
    - all other physical causes should be treated before attributing to anxiety
    - diazepam should be avoided as it may cause excessive sedation
- motion-induced nausea
    - first line is cyclizine
    - other options include hyoscine hydrobromide and cinnarizine

- nausea and vomiting of uncertain origin
    - sometimes despite investigations, sometimes because the prognosis does not warrant further investigations
    - broad spectrum antiemetic is appropriate – levomepromazine blocks 5-HT2, histamine H1 and ACh receptors

PalC6 psychosocial concerns including spiritual care and care of the family

- breaking bad news
    - find out what the patient already knows
    - find out what they want to know
        - a minority of patients may not want to know much and their right to autonomy includes this
    - follow up for patients needs to be in place so they have the chance to ask the questions they will have later
- psychological support
    - five stages of grief (Elizabeth Kübler-Ross – the five stages of receiving catastrophic news)
        - denial
            - initial stage of shock and numbness
        - anger
            - may be internalised or externalised against the clinician
            - may be difficult to deal with
            - is a mechanism for dealing with anxiety and pain
        - bargaining
        - depression
            - dying patients with depression may require treatment, particularly if experiencing morbid thoughts such as undue guilt, low self esteem and anhedonia
        - acceptance
            - there may be an intense longing for death
    - social support
        - requires consideration of:
            - the patient's social support network and the need for additional input where there is a lack of friends and relatives for support
            - personal care (e.g. toileting and bathing) needs
            - financial issues and access to local and national resources
            - respite care
    - spiritual support
        - often involve questions about fate, the purpose of life and the existence of a higher power or being
        - members of the healthcare team should be aware of the patient's spiritual needs and that they may change with time
    - support for carers
        - with patient consent, families and carers should be kept fully involved
    - co-ordinating care

        - catering for the needs of patients approaching the end of life should not be done by one single healthcare professional
        - integrated teams are most useful
        - guidelines include:
            - The Gold Standards Framework Programme England
            - The End of Life Care Programme by the Department of Health
        - organisations providing care and support include:
            - The National Council for Palliative Care
                - umbrella organisation for those involved in providing, commissioning and using palliative care and hospice services in England, Wales and Northern Ireland
            - Macmillan Cancer Support
                - gives cancer patients and their families information, practical advice and support
            - Marie Curie Cancer Care
                - charity supporting Marie Curie nurses, hospice research and information for the general public

PalC7 the dying patient

- background
    - over 60% of people express a wish to die at home
    - being pain free and the presence of family and friends are also important to many people
    - terminal care encompasses a variety of diseases including:
        - cancer
        - heart failure
        - respiratory failure
        - CKD
        - hepatic failure
        - neurological diseases e.g:
            - multiple sclerosis
            - motor neurone disease
            - dementia
- One Chance to Get it Right
    - published by the Leadership Alliance for the Care of Dying People in 2014 following a review of the Liverpool Care Pathway commissioned by the UK government
    - five 'priorities of care' form the focus of care at the end of life
        - universal and applicable irrespective of the place where the person is dying
- the possibility that the person may die within the next few hours or days is recognised and communicated clearly, decisions made and actions taken in accordance with the person's needs and wishes
    - these are regularly reviewed and decisions revised accordingly
- sensitive communication takes place between staff and the dying person and those identified as important to them
- the dying person and those identified as important to them are involved in decisions about treatment and care to the extent that the dying person wants

- the needs of families and others identified as important to the dying person are actively explored, respected and met as far as possible
- an individual plan of care, which includes food and drink, symptom control and psychological, social and spiritual support, is agreed, co-ordinated and delivered with compassion
- useful tools
    - there are several tools available to help identify people nearing the end of life, such as the Gold Standards Framework, the Amber Care Bundle or the Supportive and Palliative Care Indicators Tool (SPICT)
    - the Gold Standards Framework has a Prognostic Indicator Guide with three trigger questions:
        - would you be surprised if the patient were to die in the next months, weeks or days?
        - do they have general indicators of decline?
            - reduced ability for self care
            - repeated unplanned hospital admission
            - spending more than 50% of their time in bed
        - do they have specific clinical indicators e.g. cancer metastases, increased frequency of steroid courses for COPD?
    - identifying the 1% of the UK population who die every year allows appropriate care to be planned and benefits the individual and those close to them
- dying at home
    - adequate backup is required
        - usually a family member or team of friends
        - night sitter services are available but expensive
        - the carer must be prepared to cope with the patient's physical and emotional needs
    - team approach
        - areas that may require support include:
            - disease-specific questions, e.g.:
                - symptom management
                - hydration and nutrition
                - access to medication
            - physical problems
            - psychological problems
            - social matters, e.g. signposting advice on benefits, finance and third sector, local or national support services
            - support with activities of daily living, including access to equipment and rehabilitation services
            - pastoral, religious and spiritual guidance
            - cultural matters
        - the team may include a GP, district nurses, Macmillan nurses, Social Services and often a minister of religion
            - there should be a specific key worker who liaises with the family
                - they can ensure that nothing is missed or duplicated
        - the local provider of unscheduled care should be informed if there is likely to be a problem out of hours

- they should have a list of all terminally ill people
- unnecessary visits may be prevented, including potentially leaving certification of death until the next morning

- emotional needs
  - issues to discuss include:
    - being honest with the patient, answering their questions as best as possible, including being honest about uncertainties
    - discussions should start with signposting about asking difficult questions
      - it is important to ask whether they would like to talk about what to expect and what is likely to happen
    - the patient should make a will if they have not yet done so
- general debility
  - patients become weaker as death approaches
    - they require help in and out of bed and to the toilet
    - sleep may be excessive
    - appetite diminishes and weight falls
      - intensive nourishment may not be in the best interests of the patient
      - calorie-dense food may be poorly absorbed and cause diarrhoea and the stress of faecal incontinence
    - urinary incontinence is often followed by faecal incontinence
      - pads and waterproof undersheets are useful
      - if skin integrity breaks down it may never heal
      - a urinary catheter may be considered but requires discussion of all the options
    - immobility, poor circulation and inadequate nutrition predispose to pressure ulcers
      - frequent turning and appropriate mattresses should be considered
        - turning can be painful but necessary
- pain and distress
  - other symptoms that may be distressing include:
    - insomnia
    - anorexia
    - constipation
    - sweating
    - nausea
    - dyspnoea
    - dysphagia
    - neuropsychiatric symptoms
    - vomiting
    - urinary symptoms
    - dyspepsia
    - paresis
    - diarrhoea
    - pruritus
    - dermatological symptoms
  - considerations for management include:

        - keeping to oral medication for as long as possible
        - avoiding injections
        - consideration of patches and/or syringe drivers
        - allowing others such as the patient or carers to decide on dose increases whilst ensuring a rational regime is in place
        - appropriate spacing of medication – if doses are too far apart the patient has severe debilitating pain requiring high doses to suppress it, and spends half of the time distressed by pain and the other half sedated by heavy medication
        - in the terminal stages, some routine drugs can and should be stopped
        - sedation is frequently required and has not been shown to hasten death
- ethical issues
    - GMC guidance – 'Treatment and care towards the end of life: good practice in decision making'
    - important points include:
        - care should be equitable
            - capacity and human rights legislation should be borne in mind
        - all reasonable steps should be taken to prolong a patient's life but there is no absolute obligation to prolong life irrespective of the consequences for the patient or the patient's views
        - every adult patient should be presumed to have the capacity to make decisions about their care and treatment unless proved otherwise
        - if an adult lacks capacity, the decisions made on the patient's behalf must be based on whether treatment would be of overall benefit (including the option not to treat) in consultation with those close to the patient
        - different decision-making paths should be followed for patients who have capacity and those who do not
    - decisions about palliative sedation and the role of food and fluid intake in the prolongation of life are ethically complex
        - there are inconsistencies in approach and research is required
    - patients or relatives may request the health professional to end their life
        - Medical Assistance in Dying remains illegal in the UK
            - in places where it is legal, palliative care physicians are involved in 90% of cases
            - many palliative care bodies believe that palliation and assisted dying are incompatible
- caring for the carer
    - carers need to know what to expect, for example Cheyne-Stokes breathing
    - they need to have practical details on who to call in case of need and what to do when death occurs
        - they should not find themselves calling 999 in ignorance
    - they should be encouraged to speak to the patient about practical details such as funeral arrangements and where the will is lodged
    - they should see their GP some time after the funeral

PhP1 medication side effects/interactions

- background
    - any drug may produce unwanted or unexpected adverse reactions
    - rapid detection and reporting is important to ensure that medicines are used safely
    - healthcare professionals and coroners are urged to report suspected adverse drug reactions to the Medicines and Healthcare products Regulatory Agency (MHRA) through the Yellow Card Scheme
        - this can be done electronically at mhra.gov.uk/yellowcard or by physical cards
- yellow card scheme
    - suspected adverse drug reactions to any therapeutic agent should be reported, e.g.:
        - drugs (self-medication and prescribed medication)
        - blood products
        - vaccines
        - radiographic contrast media
        - complementary and herbal products
    - reports include suspected adverse reactions associated with misuse, overdose, medication errors or from use of unlicensed or off-label medications
    - yellow cards can also be used to report medical device incidents, defective medicines and suspected fake medicines
    - spontaneous reporting is particularly valuable for recognising possible new hazards rapidly
        - adverse reactions should be reported even if it is not certain that the drug has caused it, if the reaction is well-recognised, or if other drugs have been given at the same time
    - reports of overdoses (deliberate or accidental) can complicate assessment but provide important information on the potential toxicity of drugs
- self-reporting
    - patients and their carers can also report suspected adverse drug reactions to the MHRA through the yellow card scheme
- newer drugs and vaccines
    - the black triangle identifies newly licensed medicines that require additional monitoring by the European Medicines Agency as only limited information is available from clinical trials
    - the black triangle also appears in the patient information leaflet
    - products usually retain a black triangle for 5 years but this can be extended if required
    - for these medications, all suspected reactions, including those not considered serious, should be reported
- established drugs and vaccines
    - all suspected reactions that are serious, medically significant or result in harm should be reported
    - includes reactions that are fatal, life-threatening, disabling, incapacitating or which result in or prolong hospitalisation, or a congenital abnormality
    - examples include:

        - anaphylaxis
        - blood disorders
        - endocrine disturbances
        - effects on fertility
        - haemorrhage from any site
        - renal impairment
        - jaundice
        - ophthalmic disorders
        - severe CNS effects
        - severe skin reactions
        - reactions in pregnant women
        - drug interactions
    - for established drugs there is no need to report well-known, relatively minor side effects (e.g. dry mouth with tricyclics, constipation with opioids)
- side effects in the BNF
    - the BNF lists clinically relevant side effect for most drugs
    - an exhaustive list is not included for drugs used by specialists (e.g. cytotoxic drugs and anaesthetic drugs)
    - hypersensitivity reactions are not normally listed as they can occur with virtually all drugs
        - unless there is a specific increased risk of specific management advice
    - effects likely to have little clinical consequence such as transient rise in liver enzymes are omitted
    - side effects are generally listed alphabetically in order of frequency
    - description of frequency of side effects
        - very common
            - greater than 1 in 10
        - common
            - 1 in 100 to 1 in 10
        - uncommon
            - 1 in 1000 to 1 in 100
        - rare
            - 1 in 10,000 to 1 in 1000
        - very rare
            - less than 1 in 10,000
- prevention of adverse reactions
    - never use any drug unless there is a good indication
    - if the patient is pregnant do not use a drug unless the need for it is imperative
    - allergy and idiosyncrasy are important causes of adverse drug reactions
        - ask if the patient had previous reactions to the drug or formulation
    - ask if the patient is already taking other drugs including self-medication, health supplements, complementary and alternative therapies – interactions may occur
    - age and hepatic or renal disease may alter the metabolism or excretion of drugs so that much smaller doses may be needed
        - genetic factors may also be responsible for variations in metabolism, and therefore for the adverse effect, notably of isoniazid and the tricyclic antidepressants

- prescribe as few drugs as possible and give very clear instructions to the elderly or any patient likely to misunderstand complicated instructions
- wherever possible use a familiar drug; with a new drug be particularly alert for adverse reactions or unexpected events
- consider if excipients (e.g. colouring agents) may e contributing to the adverse reaction
    - if the reaction is minor, a trial of an alternative formulation of the same drug may be considered before abandoning the drug
- warn the patient if serious adverse reactions are likely to occur

- drug allergy (suspected or confirmed)
    - any reaction caused by a drug with clinical features compatible with an immunological mechanism
        - all drugs have the potential to cause adverse drug reactions, but not all of these are allergic in nature
    - a reaction is more likely to be caused by drug allergy if:
        - the reaction occurred while the patient was being treated with the drug or
        - the drug is known to cause this pattern of reaction or
        - the patient has had a similar reaction to the same drug or drug class previously
    - a suspected reaction is less likely to be caused by a drug allergy if there is a possible non-drug cause or if there are only gastro-intestinal symptoms present
    - the following signs, allergic patterns and timing of onset can be used to help decide whether to suspect drug allergy:
        - immediate, rapidly-evolving reactions (onset usually <1 hour after drug exposure)
            - anaphylaxis, with erythema, urticaria or angioedema, and hypotension and/or bronchospasm
            - urticaria or angioedema without systemic features
            - exacerbation of asthma e.g. with NSAIDs
        - non-immediate reactions, without systemic involvement (usually 6-10 days after first drug exposure or 3 days after second exposure
            - cutaneous reactions, e.g. widespread red macules and/or papules, or fixed drug eruption (localised inflamed skin)
        - non-immediate reactions, with systemic involvement (onset may be variable, usually 3-6 days after first drug exposure, depending on features, or 3 days after second exposure)
            - cutaneous reactions with systemic features
                - e.g. drug reaction with eosinophilia and systemic signs (DRESS) or drug hypersensitivity syndrome (DHS), characterised by widespread red macules, papules or erythroderma, fever, lymphadenopathy, liver dysfunction or eosinophilia
        - toxic epidermal necrolysis or Stevens-Johnson syndrome
        - acute generalised exanthematous pustulosis (AGEP)
    - suspected drug allergy information should be clearly and accurately documented in clinical notes and prescriptions

        - patients should be given information about which drugs and drug classes to avoid
    - referral to specialist drug allergy service
        - patients with a suspected anaphylactic reaction or a severe or non-immediate cutaneous reaction
        - patients presenting with a suspected allergic reaction or anaphylaxis to NSAIDs and local and general anaesthetics
        - patients with a suspected drug reaction or anaphylaxis associated with beta-lactam antibiotics if their condition can only be treated by beta-lactam antibiotics or they are likely to need them in the future
- oral side effect of drugs
    - oral mucosa
        - medications left in contact with or applied directly to the oral mucosa can lead to inflammation or ulceration
        - aspirin dissolved in the sulcus for toothache can lead to a white patch followed by ulceration
        - flavouring agents, particularly essential oils, may sensitise the skin
        - the oral mucosa is vulnerable to ulceration in patients treated with cytotoxic drugs (e.g. methotrexate)
        - other drugs capable of causing oral ulceration include:
            - ACEi
            - gold
            - nicorandil
            - NSAIDs
            - pancreatin
            - penicillamine
            - proguanil hydrochloride
            - protease inhibitors
        - erythema multiforme, Stevens Johnson syndrome or toxic epidermal necrolysis may be caused by a variety of drugs and can affect the oral mucosa
        - lichenoid eruptions are associated with ACEi, NSAIDs, methyldopa, chloroquine, oral antidiabetics, thiazide diuretics, gold
        - candidiasis can occur with antibacterials and immunosuppressants and occasionally corticosteroid inhalers
    - teeth and jaw
        - brown staining of teeth with chlorhexidine mouthwash, spray or gel
            - can be removed by polishing
        - iron salts in liquid form can stain the enamel black
        - rarely, superficial staining with co-amoxiclav suspension
        - intrinsic staining of the teeth is most commonly caused by tetracyclines
            - they affect the teeth if given at any time from around the fourth month in utero until the age of twelve
            - all tetracyclines can cause it
            - colour ranges from yellow to grey
        - excessive ingestion of fluoride leads to dental fluorosis with mottling of the enamel and areas of hypoplasia or pitting

        - fluoride supplements occasionally cause mild mottling (white patches) if the dose is too large for the child's age (taking into account the fluoride content of the local drinking water and toothpaste)
    - risk of osteonecrosis of the jaw is much greater for patients receiving IV bisphosphonates in the treatment of cancer than for those receiving oral bisphosphonates for osteoporosis or Paget's disease
        - all patients receiving bisphosphonates should have a dental check up and any remedial treatment before commencing treatment or as soon as possible after starting treatment
- peridontium
    - gingival overgrowth (gingival hyperplasia) is a side effect of phenytoin and sometimes of ciclosporin or nifedipine
    - thrombocytopenia may be drug related and can cause bleeding at the gingival margins (spontaneous or following mild trauma)
- salivary glands
    - the most common effect of drugs is to reduce flow (xerostomia)
    - patients with a persistently dry mouth may have poor oral hygiene and are at risk of dental caries and oral infections (particularly candidiasis)
    - implicated drugs include:
        - antimuscarinics
        - antidepressants
        - alpha-blockers
        - antihistamines
        - antipsychotics
        - baclofen
        - bupropion hydrochloride
        - clonidine hydrochloride
        - $5HT_1$ agonists
        - opioids
        - tizanidine
        - excessive use of diuretics
    - some drugs can increase saliva productions (e.g. clozapine, neostigmine) but this is rarely a problem unless the patient has difficulty in swallowing
    - pain in the salivary glands has been reported with some antihypertensives and with vinca alkaloids
    - swelling of the salivary glands can occur with iodides, antithyroid drugs, phenothiazines and sulfonamides
- taste
    - there can be decreased taste acuity or alteration in taste
    - implicated drugs include:
        - amiodarone
        - calcitonin
        - ACEi
        - carbimazole
        - clarithromycin
        - gold

- griseofulvin
- lithium salts
- metformin
- metronidazole
- penicillamine
- phenindione
- propafenone
- protease inhibitors
- terbinafine
- zopiclone

PhP2 overdose

- types of poisoning
    - deliberate
        - overdose as a self-harm or suicide attempt
        - child abuse +/- fabricated or induced illness by carers
        - third party
            - attempted homicide
            - terrorist
            - warfare
    - accidental
        - most episodes of paediatric poisoning
        - dosage area
            - iatrogenic
            - patient error
        - recreational use
        - environmental
            - plants
            - food
            - venomous bites/stings
- general management
    - advice from Toxbase or National Poisons Information Service
    - resuscitation
        - airway
            - open, suction, maintain and intubate as necessary
        - breathing
            - assess work and effectiveness of ventilation
            - give oxygen +/-assisted ventilation (avoiding mouth to mouth)
            - respiratory depression
                - consider opiates, benzodiazepines
            - tachypnoea
                - consider metabolic acidosis (e.g. salicylates, methanol)
        - circulation
            - attach cardiac monitor, assess pulse, blood pressure and perfusion
            - establish IV access
            - tachycardia/irregular pulse

        - consider:
            - salbutamol
            - antimuscarinics
            - tricyclics
            - quinine
            - phenothiazine
            - choral hydrate
            - cardiac glycosides
            - amphetamines
            - theophylline
    - if hypotensive, consider fluid bolus +/- inotropes
- disability
    - assess consciousness level (GCS or AVPU)
    - coma may suggest benzodiazepines, alcohol, opiates, tricyclics or barbiturates
    - pupils and eye movements
        - large pupils
            - anticholinergics
            - sympathomimetics
            - tricyclics
        - small pupils
            - opiates
            - cholinergics
        - unreactive
            - barbiturates
            - carbon monoxide
            - hydrogen sulphide
            - cyanide/cyanogens
            - head injury
            - hypoxia
        - unequal
            - slight variation can be normal
            - consider head injury
        - strabismus
            - can be seen with carbamazepine overdose
        - papilloedema
            - methanol
            - carbon monoxide
            - glutethimide
        - nystagmus
            - CNS acting agents (e.g. phenytoin)
    - check blood glucose
        - if hypoglycaemic give 50ml 50% dextrose IV (for children 5ml/kg 10% dextrose)
        - hyperglycaemia
            - organophosphates
            - theophyllines
            - monoamine-oxidase inhibitors

        - salicylate
    - hypoglycaemia
        - insulin
        - oral hypoglycaemics
        - alcohol
        - salicylate
- history (may be unreliable)
    - what was taken, how much and by what route
    - whether alcohol was consumed too
    - whether there has been any vomiting since ingestion
    - past medical history, current medications and allergies
    - whether a suicide note was left
    - whether the patient is pregnant
    - history from others including family, friends, paramedics, police, observers
- general examination
    - directed cardiovascular, respiratory, abdominal and neurological examination
    - vital signs
    - temperature
        - hypothermia
            - phenothiazines
            - barbiturates
            - tricyclics
        - hyperthermia
            - amphetamines
            - ecstasy
            - MAOIs
            - cocaine
            - antimuscarinics
            - theophylline
            - serotonin syndrome
        - muscle rigidity
            - ecstasy
            - amphetamines
        - skin
            - cyanosis
                - methaemoglobinemia
            - very pink
                - carboxyhaemoglobinaemia
                - cyanide
                - hydrogen sulphide
            - blisters
                - barbiturates
                - tricyclic antidepressants
                - benzodiazepines
            - needle tracks
            - hot/flushed
                - anticholinergics

        - breath
            - ketones
                - diabetic/alcoholic ketoacidosis
            - bitter almonds
                - cyanide
            - garlic-like
                - organophosphates
                - arsenic
            - rotten eggs
                - hydrogen sulphide
        - mouth
            - perioral acneiform lesions
                - solvent abuse
            - dry mouth
                - anticholinergics
            - hypersalivation
                - parasympathomimetics
- investigations
    - 12 lead ECG
    - bloods
        - U&E
        - lab glucose
        - LFT
        - CK
        - coagulation
        - VBG/ABG
            - including carboxyhaemoglobin
        - paracetamol level
        - other levels if indicated:
            - salicylate
            - theophylline
            - digoxin
            - lithium
            - antiepileptics
    - urinalysis
        - rhabdomyolysis
        - sample can be saved for possible toxicological analysis
    - CXR
        - if suspected pulmonary oedema/aspiration
    - CT brain
        - if needed to exclude other causes of altered consciousness
- differential
    - head trauma
    - stroke/subarachnoid haemorrhage
    - meningitis
    - metabolic abnormalities
        - e.g. hypoglycaemia, hyponatraemia, hypoxaemia)

    - liver disease
    - post-ictal state
- treatment
    - obtain more information
        - TOXBASE
        - National Poisons Information Centres
        - MIMS colour index or TICTAC
            - computer aided tablet and capsule identification service available to authorised users including Poisons Information Centres
        - British National Formulary/data sheet compendium
    - decontamination if appropriate
        - avoid contaminating self and wear protective clothing
        - ensure the area is well-ventilated
        - the patient should remove soiled clothing and wash themselves if possible
        - put soiled clothing in a sealed container
        - wash all contaminated skin/hair with liberal amounts of warm water/soap
    - decrease absorption
        - single dose activated charcoal
            - patient needs to have had significant overdose, be cooperative, with no impairment of consciousness and not likely to have imminent seizures
            - usual dose is 50g for an adult, 1g/kg for children
            - can be repeated after 1 hour if necessary
            - not effective for:
                - iron
                - lithium
                - boric acid
                - cyanide
                - ethanol
                - ethylene glycol
                - methanol
                - malathion
                - DDT
                - carbamate
                - hydrocarbon
                - strong acids or alkalis
            - oral antidotes given after charcoal may be ineffective
        - gastric emptying
            - contra-indicated if the airway is unprotected or an overdose of corrosives or hydrocarbons has been taken
            - complications include pulmonary aspiration and oesophageal perforation
            - can be effective within one hour of ingestion but only 30% of gastric contents are returned
                - only generally done if patients present early having taken a potentially fatal overdose

- sometimes delayed if there is delayed gastric emptying (e.g. coma or overdose of tricyclics or salicylates)
- gastric lavage may be considered for medications absorbed poorly by activated charcoal and for sustained release preparations or enteric-coated tablets
    - involves placing the patient in the left lateral head down position (20 degrees), inserting a large bore tube (36-40F in adults, 16-28F in children) into the stomach
    - contents are removed with sequential administration and aspiration of small quantities of warm water or saline (200-300ml in adults, 10-20ml/kg in children, preferably saline)
    - alternatively the stomach contents can just be aspirated

▪ whole bowel irrigation
- may be considered in cases where substances would not be absorbed by activated charcoal
- uses large volume of osmotically balanced, non-absorbable polyethylene glycol electrolyte solution (e.g. Klean-Prep)
- used with iron and other heavy metals, lithium, sustained-release or enteric coated products, large ingestions and ingested drug packets
- administered at 1-2L/hr (30ml/kg hour in children) PO or via NG
- antiemetics may be required
- should be continued until rectal effluent is clear (around 3-6 hours)
- rarely used

o increase elimination

▪ multiple doses of activated charcoal
- interrupts enterohepatic or enteroenteric recirculation
- 50g four hourly or 12.5g hourly (children 1g/kg or 0.25g/kg)
- severe constipation and fluid depletion can occur
- used with:
    - carbamazepine
    - dapsone
    - phenobarbital
    - quinine
    - salicylate
    - colchicine
    - dextropropoxyphene
    - digoxin
    - verapamil
    - theophylline

▪ forced diuresis
- no longer recommended

▪ haemoperfusion and acid/alkaline diuresis
- rarely used

▪ haemodialysis
- severe salicylate, ethylene glycol, methanol, lithium, phenobarbital, chlorate

o supportive

        - maintain ABCDs
        - observation and treatment of late complications (e.g. liver failure, rhabdomyolysis)
    - specific antidotes
- psychiatric assessment
    - intentions at the time
        - whether the act was planned
        - what precautions were taken against being found
        - whether the patient sought help afterwards
        - whether the patient thinks the method was dangerous
        - whether there was a final act such as a suicide note
    - problems which led to the act
        - whether they still exist
        - whether the act was aimed at someone
        - whether there is a psychiatric disorder (e.g. depression, alcoholism, personality disorder, schizophrenia, dementia)
    - what resources they have
        - friends
        - family
        - work
        - personality
    - present intentions and suicide risk
        - factors increasing the chance of future suicide:
            - original intention was to die
            - present intention is to die
            - presence of psychiatric disorder
            - poor resources
            - previous suicide attempts
            - socially isolated
            - unemployed
            - male
            - age over 50
- prevention
    - adult education
    - double checking of dosage before administration
    - vigilance by healthcare professionals to recognise the early signs of abuse and potential suicide
    - all medicines and household chemicals in a locked childproof cupboard >1.5 metres off the ground
    - safe disposal of medicines and chemicals which are not needed or are out of date
    - all medicines and chemicals should be kept in their original containers with clear labels

PhP3 accidental poisoning

- paediatric toxicology
    - background

        - presentation of a child to the ED after ingestion of a potential toxin is common
        - there are around 40,000 attendances per year for suspected poisoning in children in England and Wales per year
            - about half are admitted for treatment or observation
        - the majority of paediatric ingestions involve accidental unintentional exposure to small doses of non-toxic or minimally toxic substances in children between the ages of one and five
            - it can often be difficult to establish whether or not an ingestion has actually occurred
        - more than 80% of ingestions occur in the home
        - incidence is partly related to availability
        - around 40% relate to ingestion of pharmaceutical preparations
            - most commonly iron, tricyclic antidepressants, benzodiazepines, paracetamol and oral contraceptive pills
        - in the majority of cases, a brief period of observation and parental reassurance is all that is required
            - less than 1% are clinically serious and death is rare
        - child resistant containers have helped to reduce incidence, but a significant proportion of children under 5 can open them
    - child protection
        - safeguarding must always be considered for any paediatric toxicological presentation
        - of children under 6 with accidental ingestion, 30% have a further episode
            - there is also potential harm for other members of the household
            - the family network may require further support
        - poison prevention advice and education should be provided and further follow-up arranged via a health visitor or school nurse referral
        - if there is significant risk or concerns about deliberate poisoning, the case will need to be discussed with the Child Protection lead
    - types of paediatric poisoning
        - accidental poisoning
            - most frequent
            - generally in under 5s with boys under 2 the commonest group
            - often occurs when there is inadequate supervision of the child and psychosocial stress within the family (e.g. new baby, maternal depression, substance misuse)
            - personality traits of the child such as hyperactivity can be important
        - deliberate self-poisoning
            - generally involves teenagers and reflects adult patterns of self-poisoning
            - includes teenagers and young adults experimenting with alcohol and drugs
            - they require assessment by age-appropriate psychiatric and social teams
        - iatrogenic poisoning

        - prescribing is more hazardous due to differences in size, body surface area and immaturity of metabolic pathways
        - drug calculations should always be double-checked
    - deliberate poisoning
        - fabricated or induced illness by proxy
        - rare, but should be considered in children presenting with an unclear constellation of symptoms
            - doctors may or may not have social concerns for the child
        - substances used include salt, emetics and purgatives
- differences with adults
    - anatomical differences
        - body weight
            - can vary considerably with age and nutritional status
        - body surface area
            - larger in children than in adults so there is a larger absorptive area for agents absorbed through the skin
            - sometimes used instead of weight to calculate drug doses
            - the skin of children is also thinner and the keratinised epithelium less well developed
        - airway calibre
            - much smaller than in adults
            - any further reduction due to constriction (bronchospasm) or obstruction (secretions or gastric contents) has a significant impact (to the fourth power as per Poiseuille's law)
                - e.g. halving the radius causes air flow to fall one sixteenth
        - body water
            - proportionally greater than in children resulting in different volumes of distribution
    - physiological differences
        - ventilation
            - much greater minute volume than adults
            - more susceptible to the effects of gases, vapours and aerosolised agents (carbon monoxide)
        - cardiac output
            - children increase cardiac output primarily by mounting a tachycardia and have limited capacity to increase stroke volume
            - they can decompensate quickly if they lose fluid or become bradycardic (e.g. with organophosphorus compounds)
        - renal and liver function
            - immature function predisposes to impaired ability to eliminate and detoxify toxic substances
        - glycogen stores
            - lower glycogen stores within the liver predisposes to early hypoglycaemia when they experience a physiological stress (e.g. seizures or sepsis)

- blood brain barrier
    - greater permeability allows toxic agents greater potential to gain entry into the central nervous system
    - the CNS is also still developing and therefore at risk from certain exposures (e.g. lead) and secondary brain injuries
- neonates
    - some agents ingested by the mother in pregnancy can cross the placenta and lead to dependence in the foetus which can present as withdrawal after birth
    - these include:
        - amphetamines
        - barbiturates
        - caffeine
        - cocaine
        - opioids
        - SSRIs
    - toxic agents may also be transferred in breast milk (e.g. aspirin, cocaine, lithium)

- developmental considerations
    - height
        - children have access to areas that adults may overlook (e.g. under sink storage)
    - developmental milestones
        - children aged 1-5 are adventurous by nature
        - they do not always appreciate danger and tend to copy their parents
            - particularly important when medicines are taken in their sight
        - when considering safeguarding it is important to consider whether mechanism of ingestion is consistent with the developmental age
            - ingestions under the age of one should be carefully assessed before being deemed accidental

- history
    - what was ingested
        - any empty packets, unconsumed pills, berries, leaves or their descriptions
        - attempt to establish potential toxins consumed
    - maximum possible amount ingested
        - should be calculated on a mg/kg basis after weighing the child
        - where more than one child is involved assume each has consumed the maximum possible dose (worst case scenario)
    - other toxins available
        - which prescription medications are available in the household or other household products present
        - particularly important for poisoning from an unknown substance

        - consider plant or fungi ingestion in an acutely unwell child who has been outside
    - when the ingestion occurred
        - aim to establish time of ingestion (may be a time frame)
    - symptoms since possible ingestion
        - specifically coughing, choking, vomiting, diarrhoea, reduced level of consciousness
    - whether it could be a deliberate ingestion
        - these will need formal psychiatric assessment before discharge
- one pill can kill list (in children <10kg)
    - tricyclic antidepressants (dothiepin)
    - chloroquine and hydroxychloroquine
    - calcium channel blockers (diltiazem, verapamil)
    - opioids (oxycodone, methadone, morphine sulphate controlled release)
    - amphetamines (amphetamine, MDMA)
    - propranolol
    - theophylline
    - sulphonylureas (gliclazide, glibenclamide)
    - other toxic substances:
        - camphor (Vicks Vaporub, tiger balm)
        - methylsalicylate (Oil of Wintergreen)
        - toxic alcohols (methanol, ethylene glycol)
        - iron supplements
        - essential oils
- examination
    - likely to be normal in a child presenting to the ED soon after an ingestion
        - may however provide information about the nature of an unknown toxin
    - vital signs
        - take seriously observations outside the normal range for the age of the child
        - include temperature
            - hyperpyrexia
                - ecstasy
                - cocaine
                - salicylates
            - hypothermia
                - ethanol
                - barbiturates
    - mental status
        - AVPU score usually used in children
    - neurological examination
        - neurological signs affecting one side of the body should not be attributed to poisoning
        - looks for external signs of head injury or other evidence of non-accidental injury
    - eyes, mucous membranes, skin, bowels, bladder

- may suggest a toxidrome
- significant toxidromes include:
    - opiate
    - sympathomimetic
    - salicylism
    - serotinergic
    - anticholinergic (e.g. tricyclics, antihistamines, deadly nightshade)
    - cholinergic (organophosphorus and carbamate compounds)
- if more than one substance has been taken, this may not be clear

▪ exclude alternative causes
- other organic conditions may present in a similar way
- the clinician should also have a low threshold for considering a toxicological cause for presentations where a medical cause cannot be found

o investigations
▪ not required in most cases with non-toxic ingestions
▪ blood glucose
- should be a consideration as it is easily remedied
- mandatory for altered mental status
- no requirement for repeat measurements if the first is normal unless the toxin is known to cause hypoglycaemia or the child deteriorates

▪ ECG
- 12 lead mandatory if the substance is known to cause arrhythmias
- also for any child with altered mental status or abnormal heart rate or blood pressure
- interpretation is different to adult ECGs

▪ blood gas analysis
- capillary gas may be taken to assess acid-base status
- can check sodium if deliberate poisoning suspected

▪ paracetamol
- less common than in adults and should not be routinely checked except where there is concern about access to paracetamol by the child
- tends to occur in deliberate self-harm and should then be checked

▪ x-rays
- may be considered for ingestion of radio-opaque substances or foreign bodies
- COINS mnemonic
    - **C**hloral hydrate, calcium
    - **O**piate or other drug packets
    - **I**ron and other metals
    - **N**euroleptic agents
    - **S**ustained release or enteric coated preparations
- button batteries

        - can result in localised mucosal ulceration with risk of GI haemorrhage or perforation if they remain in the oesophagus or stomach
        - once the battery has passed the pylorus it is unlikely to cause toxicity
    - magnets
        - when one or more magnets are ingested there is a risk of them becoming adherent with intervening bowel tissue which can become eroded or perforate and cause bleeding
- urine testing
    - qualitative bedside testing strips unlikely to be helpful
        - accidental ingestions are not usually of the drugs tested
        - in adolescents it is unlikely to change management
    - if there is a suspicion of deliberate poisoning, urine samples may be used for forensic purposes but chain of custody would be required
- management
    - risk assessment after history and examination
    - management is largely supportive
    - if the substance is low or non-toxic as per Toxbase, reassurance can be given with advice to return if symptoms such as vomiting, rash or altered conscious level develop
    - unknown ingestion
        - NPIS have access to TICTAC for drug identification and to the Royal Botanical Gardens at Kew for plant identification
        - general management
            - good supportive care and appropriate observation
            - aim for normothermia, euglycaemia, euvolaemia and prompt management of seizures
            - benzodiazepines are first line for seizures
                - phenytoin should be avoided due to sodium channel blocking properties
            - activated charcoal may be appropriate in a small number of cases
                - it can be mixed with a cola-type drink to make it more palatable
        - specific therapies
            - antidotes
                - rarely required in paediatric poisonings
                - should not distract from good supportive care
            - elimination for aspirin toxicity
                - may include urinary alkalinisation +/- haemodialysis

PhC1 overdose of prescription and non-prescription medications including legal and non-legal drugs

- paracetamol overdose
    - background
        - one of the most common agents of intentional self-harm

        - most common cause of acute liver failure
        - purchase is limited to16 tablets (32 in pharmacies) in the UK
    - toxicity
        - risk of severe liver damage (peak ALT >1000 IU/L)
            - less than 150mg/kg – unlikely
            - more than 250mg/kg – likely
            - more than 12g total – potentially fatal
        - there is however considerable variability including based on age, health and co-ingestion
    - pathophysiology
        - paracetamol is absorbed well from the stomach and small intestine after oral ingestion
        - peak plasma concentration is usually reached within an hour (or 30 minutes if in liquid or rapidly absorbed form)
        - mainly inactivated by the liver by conjugation leading to two metabolites – glucuronide or sulphate, then renally excreted through urine
            - in overdose the liver conjugation pathway becomes inundated, causing paracetamol to be metabolised by an alternative pathway
            - results in a toxic metabolite, NAPQI, which is inactivated by glutathione
            - when glutathione stores are depleted to less than around 30%, NAPQI reacts with nucleophilic aspects of the cell, leading to necrosis
                - necrosis occurs in the liver and the kidney tubules
        - risk is possibly increased with:
            - induction of the P450 system (e.g. rifampicin, phenytoin, carbamazepine, alcohol)
            - genetic variation
            - HIV positive status
            - malnutrition
            - alcohol-related or other liver disease
        - paediatric patients under 5 tend to fare better but should be treated the same due to lack of evidence
    - clinical features
        - commonly asymptomatic for first 24 hours or non-specific symptoms such as nausea and vomiting
        - hepatic necrosis begins to develop after 24 hours
            - elevated transaminases
            - right upper quadrant pain
            - jaundice
            - can progress to acute liver failure
        - patients may also develop:
            - encephalopathy
            - oliguria
            - hypoglycaemia
            - renal failure - usually occurs around day 3
            - lactic acidosis

- history
  - number of tablets, formulation, any concomitant tablets (including herbal substances such as St John's wort)
  - time of overdose
  - whether a suicide note was left
  - any alcohol taken
    - acute ingestion may reduce NAPQI production
    - chronic alcoholism may increase it (theoretical rather than evidence based)
- examination
  - usually normal until acute liver failure develops
  - in acute liver failure:
    - jaundice
    - hepatic flap
    - encephalopathy
    - tender hepatomegaly
- investigations
  - paracetamol level
    - at four hours after ingestion
    - on arrival if greater than four hours or staggered (levels not helpful in staggered overdose except to confirm ingestion)
  - U&Es
    - for baseline and to check for renal failure
  - LFTs
    - may be normal if presenting early
    - ALT >100 IU/L indicates hepatotoxicity
  - glucose
    - capillary glucose should be checked hourly in hepatic necrosis as hypoglycaemia is common
  - clotting screen
    - prothrombin time is the best indicator of severity of liver failure
    - INR should be checked 12 hourly
  - ABG
    - acidosis can occur at a very early stage even when the patient is asymptomatic
    - seen in 10% of patients with acute liver failure
  - FBC and salicylate levels are not routinely required
- management
  - the nomogram for treatment is ultra-conservative and there is a lack of consensus on management internationally
  - patients with a timed level on or above the treatment line should be treated
  - if there is doubt about timing, including a staggered overdose over one hour or more, treatment should be started immediately
  - discussion with expert for:
    - modified-release paracetamol
    - IV paracetamol
    - massive overdose (>1g/kg)

        - multiple drug overdose
    - ICU referral for fulminant liver failure
    - N-acetylcysteine treatment
        - acts as a precursor for glutathione
        - supplies thiols that act as antioxidants
        - virtually 100% effective in preventing liver damage when given within 8 hours of ingestion
            - efficacy decreases sharply after 8 hours
        - three consecutive infusions are given:
            - 150mg/kg over 1 hour
            - 50mg/kg over 4 hours
            - 100mg/kg over 16 hours
        - should be a total dose of 300mg/kg over 21 hours
            - a ceiling weight of 110kg should be used when calculating the dose for obese patients
            - continued treatment at the dose and rate of the third infusion may be required (usually if blood tests are still significantly abnormal)
- late presentation
    - for patients presenting >24 hours after ingestion – management is controversial and should be guided by Toxbase
    - check INR, creatinine, ALT and venous acid-base balance or bicarbonate
        - discuss with NPIS if any are abnormal
    - plasma level of paracetamol after 24 hours is likely to be undetectable even after significant overdose
        - a detectable level suggests a very large overdose, a mistake in timing of ingestion or a staggered overdose
    - a full dose of antidote therapy should be given if paracetamol is detected
- criteria for referral to specialist units
    - encephalopathy or raised ICP
        - signs of CNS oedema include sustained BP >160/90, brief rises to systolic >200, bradycardia, decerebrate posture, extensor spasms, poor pupil responses
    - INR >2.0 at or before 48 hours or >3.5 before 72 hours
        - peak elevation is at 72-96 hours
        - LFTs are not good markers of hepatocyte death
    - renal impairment (creatinine >200)
        - urine flow and daily U&Es should be measured
    - blood pH <7.3
    - systolic BP <80 despite adequate fluid resuscitation
    - hypoglycaemia
    - metabolic acidosis (pH <7.3 or bicarbonate <18 mmol/L)
- prognosis
    - mortality from severe liver failure is <5% with good supportive care
    - transplantation has only limited application but patients must be identified as early as possible, preferably on the second day
    - indicators for poor prognosis include:

- arterial pH <7.30 on or after day 2
- combination of prothrombin time >100 seconds, creatinine >300 and grade 3 or 4 hepatic encephalopathy
- increase in prothrombin time between day 3 and 4

- salicylates
  - background
    - potentially fatal
    - found in aspirin medications and high concentrations of Oil of Wintergreen (methyl salicylate – 1 teaspoon = 7g salicylate)
    - children and adults who have ingested <125mg/kg aspirin and have no symptoms do not require admission
      - ingestion of >250mg/kg is likely to cause moderate toxicity and >500mg/kg causes severe toxicity and possibly death
  - risk factors for death
    - children and the elderly
    - late presentation
    - pulmonary oedema, CNS features, hyperpyrexia
    - metabolic acidosis
    - salicylate concentration above 700 mg/L (5.1 mmol/L)
  - presentation
    - mild poisoning
      - nausea
      - vomiting
      - tinnitus
      - lethargy
      - dizziness
    - more severe poisoning
      - dehydration
      - restlessness
      - sweating
      - warm extremities with bounding pulses
      - increased respiratory rate
      - hyperventilation
      - deafness
    - acid-base balance disturbance is normally present
    - hyperglycaemia, normoglycaemia or hypoglycaemia (all with intracellular glucose depletion) may occur
    - uncommon features
      - haematemesis
      - hypokalaemia
      - hyponatraemia/hypernatraemia
      - hypocalcaemia
      - thrombocytopenia
      - abnormal coagulation (increased prothrombin ratio/INR
      - DIC
      - AKI
      - non-cardiac pulmonary oedema

        - CNS: confusion, disorientation, coma, convulsions (less common in adults than children)
    - chronic salicylate poisoning
        - more common in the elderly, requires lower levels of ingestion
        - often goes unrecognised
        - onset may be insidious
        - features may include:
            - anxiety
            - tachypnoea
            - diffuse sweating
            - difficulty concentrating
            - confusion
            - hallucinations
            - agitated delirium
            - elderly patients may present with a decline in functional status
        - salicylate poisoning should be considered in the differential for an adult with unexplained acid-base abnormality, especially if there are neurological symptoms
        - delayed diagnosis results in increased morbidity and mortality
    - investigations
        - plasma salicylate concentrations
            - the severity of poisoning cannot be assessed by levels alone and the clinical and biochemical picture must be assessed
            - should be measured urgently for patients suspected of taking >125mg/kg aspirin or those who have taken any other form
            - should be taken at least two hours post-ingestion for symptomatic patients and four hours for asymptomatic patients
            - a repeat sample should be taken 2 hours later because of the possibility of continuing absorption
                - measurements should be repeated every 3 hours until levels are falling
        - other bloods
            - U&Es
                - plasma potassium should be checked every 3 hours and levels maintained at 4.0-4.5 mmol/L
            - FBC
            - coagulation
            - glucose
        - urinary pH
        - ABG (or VBG/capillary gas in children)
            - in adults and children over 4: mixed respiratory alkalosis and metabolic acidosis, with normal or high arterial pH
            - young children: metabolic acidosis is common
    - likelihood of toxicity
        - ingested dose
            - >125mg/kg – likely toxicity mild
            - >250mg/kg – likely toxicity moderate

- >500mg/kg – likely toxicity severe, possibly fatal
- salicylate concentration
  - most adult deaths occur in patients with concentrations >700mg/L
- clinical grading
  - mild (nausea, vomiting, tinnitus)
  - moderate (hyperventilation and confusion)
  - severe (hallucinations, seizures, coma, cerebral oedema, pulmonary oedema
- acid-base
  - stage I: blood pH >7.4, urine pH >6.0
    - respiratory alkalosis, increased urinary excretion of bicarbonate
  - stage 2: blood pH >7.4, urine pH <6.0
    - metabolic acidosis with compensating respiratory alkalosis, urinary hydrogen excretion, intracellular potassium depletion
  - stage III: blood pH <7.4, urine pH <6.0
    - severe metabolic acidosis and hypokalaemia

- management
  - activated charcoal if toxic dose and presenting within an hour
    - second dose may be required if plasma levels continue to rise or the aspirin was enteric coated
  - consideration of gastric lavage if patient has ingested >500mg/kg and presents within an hour
  - aggressive rehydration
  - low threshold to give glucose – intracellular depletion may not be reflected in the blood level
  - urinary alkalinisation
    - may increase elimination
    - optimal pH is 7.5-8.5
    - if concentration >500 mg/L in adults, 225 mmol sodium bicarbonate (225ml 8.4% over 60 minutes or 1.5L 1.26% over 2 hours)
    - if concentration >350 mg/L in a child, 1ml/kg 8.4% bicarbonate diluted in 0.5L 5% dextrose at 2-3ml/kg/hr
    - urinary pH should be checked hourly
      - further doses may be required
    - does not need to be delayed whilst awaiting dialysis but volume overload must be avoided in an oliguric patient
    - hypokalaemia should be corrected before giving sodium bicarbonate – it may make urinary alkalinisation less effective
  - forced diuresis should not be used
  - haemodialysis
    - treatment of choice for severe poisoning
    - should be considered for:
      - plasma concentrations >700mg/L
      - AKI
      - congestive cardiac failure

        - non-cardiogenic pulmonary oedema
        - coma
        - convulsions
        - CNS effects not resolved by correction of acidosis
        - persistently high salicylate concentrations unresponsive to urinary alkalinisation
        - severe metabolic acidosis (pH <7.2)
      - patients <10 or >70 may require dialysis at an earlier stage
      - haemofiltration is less efficient but may be an option in hospitals without dialysis facilities
    - mechanical ventilation
      - may be indicated for deteriorating mental status or acute lung injury or uncontrollable agitation
      - can cause rapid worsening of clinical toxicity unless normal or slightly alkalaemic blood pH is maintained by hyperventilation (reducing CO_2) and/or IV sodium bicarbonate

- SSRIs
  - background
    - common in overdose, usually follow a benign course
    - serotonin toxicity occurs in a minority
      - more common with co-ingesting agents with serotonergic action
    - citalopram (>600mg) and escitalopram (>300mg) are unique in their ability to cause dose dependent QT prolongation and risk of torsades de points
  - toxic mechanism
    - enhancement of central serotonergic neurotransmission by inhibiting serotonin reuptake
  - resuscitation
    - seizures
      - IV benzodiazepines titrated every 5 minutes to effect
      - check the patient is not in a dysrhythmia
      - generally 0.1mg/kg lorazepam (max 4mg)
      - occur in <4% and more likely with citalopram
    - torsades
      - very rare
    - serotonin toxicity
      - usually mild symptoms
      - in 20% of patients
      - usually lasts <12 hours
  - supportive care
    - agitation and tachycardia
      - may herald the onset of seizures
      - titrated doses of benzodiazepines until gentle sedation achieved and heart rate falls towards 100bpm
  - investigations
    - 12 lead ECG
    - glucose
    - paracetamol levels

        - continuous cardiac monitoring if risk of torsades
            - QT interval should be measured on the nomogram not using the Bazett formula
            - if only one lead is to be used, V2 is the most accurate
    - decontamination
        - if >600mg citalopram or 300mg escitalopram ingested, 50g activated charcoal can be given within 4 hours (weighed against risk of seizure and aspiration)
            - overdose with other SSRIs does not require charcoal
    - antidotes
        - for serotonin toxicity:
            - cyproheptadine
                - antihistamine with anti-serotonergic effects
                - given orally or NG
                - 8mg, given TDS for 24 hours if positive response after first dose
            - olanzapine
                - 5-10mg sublingual
    - disposition
        - most children can be discharged unless symptomatic
        - 8 hours monitoring for citalopram >600mg and escitalopram >300mg
        - at least 12 hours monitoring for citalopram >100mg and escitalopram >500mg
        - all other patients require 6 hours of observation and are medically cleared at the end of this if they have a normal 12 lead ECG and are asymptomatic
        - symptomatic patients require 12-24 hours of supportive care or HDU/ICU care if severe
- tricyclic overdose
    - features (rapid onset, within 1-2 hours)
        - sedation and coma
        - seizures
        - hypotension
        - tachycardia
        - broad complex dysrthythmias
        - anticholinergic syndrome (tachycardia, mydriasis, dry mouth)
    - ECG
        - QRS >100ms is predictive of seizures
        - QRS >160ms is predictive of ventricular arrhythmias
    - management of significant overdose (>10mg/kg with ECG changes)
        - resuscitation room
        - IV access, high flow oxygen, monitoring
        - IV sodium bicarbonate every few minutes until BP improves and QRS narrows
        - intubate as soon as possible and hyperventilate to maintain pH 7.50-7.55
        - NG tube and activated charcoal once airway secured
        - treat seizures with IV benzodiazepines
        - treat hypotension with crystalloid bolus (10-20ml/kg

                - if unsuccessful may need vasopressors such as noradrenaline infusion
            - if arrhythmias occur, repeat sodium bicarbonate
                - lidocaine 1.5mg/kg IV is third line after sodium bicarbonate and hyperventilation, once pH is >7.5
            - avoid procainamide, flecainide, beta-blockers and amiodarone – may worsen hypotension and conduction abnormalities
            - admit to intensive care
- iron overdose
    - background
        - can have local gastrointestinal effects as well as systemic toxicity
        - risk assessment is based on the amount of elemental iron ingested
        - iron does not bind to activated charcoal
            - endoscopic/surgical decontamination may be appropriate
            - whole bowel irrigation has been used
        - a specific antidote, desferrioxamine, is available
    - mechanism of toxicity
        - local effects
            - corrosive injury to the gastrointestinal mucosa resulting in vomiting, diarrhoea, haematemesis, melaena and fluid losses that may result in hypovolaemia
        - systemic effects
            - exact mechanism unknown
            - iron acts as a cellular toxin targeting the cardiovascular system and the liver, with secondary CNS effects, metabolic acidosis due to hyperlactaemia and free proton production from the hydration of free ferric ions, and coagulopathy
    - risk assessment
        - according to dose of elemental iron
            - <20mg/kg – asymptomatic
            - 20-60mg/kg – GI symptoms only
            - 60-120mg/kg – potential for systemic toxicity
            - >120mg/kg – potentially lethal
        - for elemental iron dose:
            - ferrous sulphate – divide dose by 3.3
            - ferrous sulphate – divide dose by 5
            - ferrous gluconate – divide dose by 9
            - ferrous fumarate – divide dose by 3
    - clinical features
        - usually goes through 5 stages, which can overlap
            - 0-6 hours
                - vomiting, diarrhoea, haematemesis, melaena, abdominal pain
                - significant fluid losses may lead to hypovolaemic shock
            - 6-12 hours
                - gastrointestinal symptoms wane and the patient appears to be getting better

        - iron shifts intracellularly from the circulation
    - 12-48 hours
        - cellular toxicity becomes manifest as vasodilative shock and third-spacing, high anion gap metabolic acidosis and hepatorenal failure
    - 2-5 days
        - acute hepatic failure – rare but with high mortality
    - 2-6 weeks
        - chronic sequelae in survivors
            - cirrhosis, gastrointestinal scarring, strictures
- investigations
    - blood glucose
    - ECG
    - paracetamol
    - serum iron concentration
        - peak levels at 4-6 hours after ingestion
        - levels fall after 6 hours due to intracellular shift
        - levels do not correlate clearly with clinical toxicity
            - >90μmol/L is generally considered predictive of systemic toxicity
    - blood gas
        - presence of high anion gap metabolic acidosis is a useful marker of systemic toxicity
        - in the absence of iron levels, serum bicarbonate can be used a surrogate marker
    - abdominal x-ray
        - may confirm ingestion
    - LFTs, coagulation
        - hepatic failure
    - U&Es
        - renal failure
- management
    - resuscitation
        - ABCs
        - restoration of circulating volume
            - boluses of 10-20ml/kg crystalloid to prevent shock from GI loss, vasodilation and third spacing
    - supportive care and monitoring
    - decontamination
        - whole bowel irrigation can be considered for confirmed ingestions >60mg/kg
        - surgical or endoscopic removal of tablets if lethal ingestion (e.g. >120mg/kg)
    - antidote
        - desferrioxamine chelation in cases of systemic toxicity
        - indications:
            - level >90μmol at 4-6 hours post ingestion

        - evidence of systemic toxicity
        - shock
        - metabolic acidosis
        - altered mental status
    - mechanism
        - chelates free ferric and ferrous ions in the plasma resulting in water soluble complexes that can be renally excreted
        - removes iron bound to transferrin and haemosiderin in the vascular compartment (not from stores in other compartments)
        - ferrioxamine is excreted unchanged in the urine, which turns reddish
    - administration
        - IV infusion 15mg/kg/h, reduced if hypotension occurs, may be titrated up to 40mg/kg/h in severe toxicity
        - mandatory cardiac monitoring
    - adverse effects
        - hypersensitivity
        - hypotension (rapid or high dose IV administration)
        - ARDS (with infusions >24h)
        - toxic retinopathy
        - Yersinia sepsis (the ferrioxamine complex is a siderophore that promotes growth)
    - usually <24 hours of treatment required, can be stopped when clinically stable and serum iron <60μmol/L
- disposition
    - home if asymptomatic at 6 hours with negative abdominal x-ray
    - admit if symptomatic
        - may require IV fluids
    - patients with potential for systemic toxicity should be managed at hospitals where iron levels can be measured and chelation therapy administered if required
- calcium channel blockers (e.g. verapamil, diltiazem)
    - background
        - all deliberate self-poisonings should be considered potentially lethal
        - serious toxicity can result from >10 tablets in adults, or 1-2 SR tablets in children
    - clinical features
        - symptom onset generally 1-2 hours for standard preparations
            - may be delayed 12-16 hours with slow release preparations
        - early signs include:
            - bradycardia
            - first degree heart block
            - hypotension
            - may progress to refractory shock and death without appropriate intervention

- complications may include myocardial ischaemia, stroke and non-occlusive mesenteric ischaemia
- seizures and coma are rare and usually suggest co-ingestant
  - can occur as a late feature due to cardiovascular collapse

- effects
  - cardiotoxicity
    - negative inotropy – myocardial depression
    - negative chronotropy – sinus bradycardia
    - negative dromotropy – atrioventricular node blockade
    - effects on vascular smooth muscle result in:
      - decreased afterload
      - systemic hypotension
      - coronary vasodilation
  - metabolic effects
    - hyperglycaemia due to hypoinsulinaemia and insulin resistance
- decontamination
  - activated charcoal if presenting within 1 hour for standard release, 4 hours for slow release
  - whole bowel irrigation can be considered if the patient is cooperative, presents within 4 hours of ingestion of >10 tablets and there is no evidence of established toxicity
- management
  - fluid resuscitation
    - up to 20mg/kg crystalloid
  - calcium
    - useful temporising measure to increase HR and BP
    - options:
      - 10% calcium gluconate 60ml IV (0.6-1.0ml/kg in children)
      - 10% calcium chloride 20ml IV (0.2ml/kg in children)
        - should be given via central venous access
    - repeat boluses up to 3 times
    - consider infusion to keep serum calcium >2.0 mEq/L
  - atropine
    - 0.6mg every 2 minutes up to 1.8mg
  - catecholamine infusions
    - titrated to effect
    - e.g. dopamine, adrenaline, noradrenaline
  - sodium bicarbonate
    - consider in severe metabolic acidosis
  - cardiac pacing
    - electrical capture may be difficult to achieve and may not improve overall perfusion
- high doe insulin euglycaemic therapy
  - start with:
    - 50ml 50% dextrose IV bolus (unless marked hyperglycaemia – glucose >22 mmol/L)

                - short acting insulin 1 IU/kg bolus to maximally saturate insulin receptors
            - continue with:
                - short acting insulin infusion starting at 0.5 IU/kg/h and titrated every 30 minutes to maximum 5 IU/kg/h
                - dextrose 25g/h IV infusion to maintain euglycaemia (5.5-14 mmol/L)
                    - central venous access may be required to allow use of concentrated solutions (e.g. 50% dextrose) and limit excess volume
            - monitor:
                - glucose every 20 minutes for the first hour, then every hour
                - potassium – replace if <2.5 mmol/L and there is a source of potassium loss
                    - may actually be beneficial by augmenting myocardial contractility by enhancing calcium entry during systole, and by allowing increased intracellular potassium to have a membrane-stabilising effect in excitable cells
            - therapeutic end points:
                - improvement in myocardial ejection fraction, increased BP (systolic >90)
                - adequate heart rate (>60)
                - resolution of acidaemia; euglycaemia; adequate urine output (1-2ml/kg/h)
                - reversal of cardiac conduction abnormalities (QRS <120ms)
                - improved mentation
            - therapy is weaned after the withdrawal of vasopressors, and dextrose may be required after cessation of insulin
            - adverse effects can include:
                - hypoglycaemia
                - hypokalaemia
                - hypomagnesaemia
                - hypophosphataemia
            - adverse effects are predictable, uncommon and easily managed and the therapy is safe
- non-legal drugs
    - cocaine
        - background
            - classic sympathomimetic, known for appetite suppressant and analgesic properties
            - ingestion of >1g is potentially lethal
                - an average line is 20-30mg
            - cocaine should be on the ‘one pill can kill’ list for children
            - life threats include:
                - hyperthermia
                - hypertension
                - dysrhythmia
                - ischaemia

        - seizures
        - dissections
        - intracerebral haemorrhage
        - cerebral oedema
    - management involves:
        - benzodiazepines
            - agitation, hypertension, tachycardia, hyperthermia
        - sodium bicarbonate
            - dysrhythmias
                - can use lidocaine if refractory
        - beta blockers are contraindicated and thrombolysis is relatively contraindicated
- complications to consider and exclude
    - CNS
        - agitation
        - aggression
        - psychosis
        - myoclonic movements
        - seizures
        - intracerebral bleeds
        - cerebral oedema
    - CVS
        - tachycardia
        - dysrhythmias
        - acute coronary syndrome
        - acute pulmonary oedema
        - aortic or carotid dissection
        - ischaemic colitis
    - other
        - hyperthermia
        - pneumothorax
        - rhabdomyolysis
        - pneumomediastinum
- ventricular tachycardia
    - 50-100mmol sodium bicarbonate
    - defibrillation is used as per protocol but may not be successful due to sodium channel blockade
    - if unsuccessful, 1.5mg/kg of IV lidocaine followed by an infusion of 2mg/min
        - lidocaine is also a sodium channel blocker – there is a theory that it acts as a competitive inhibitor at a receptor level but also interacts less with the receptors leaving them free to work as a sodium channel
- acute coronary syndrome
    - beta blockers are contraindicated
    - management includes:
        - aspirin (and other antiplatelets as per hospital policy)

        - nitroglycerine
        - calcium channel antagonists
        - coronary angiography +/- stenting
    - thrombolysis contraindicated if:
        - severe hypertension
        - seizures
        - intracranial haemorrhage
        - aortic dissection
    - CT brain
        - should be considered to help weight up risk of anticoagulation
        - should be considered even if angiography planned as anticoagulation likely
- SVT
    - benzodiazepines
    - verapamil 5mg IV
    - adenosine 6-12mg IV
    - cardioversion if unstable
- hypertension
    - benzodiazepines first line
    - if refractory:
        - phentolamine 1mg IV every 5 minutes
            - used in theatre for phaeochromocytoma to control blood pressure
            - reversible non-selective alpha-adrenergic antagonist; causes vasodilatation due to alpha 1 blockade
        - vasodilator infusion such as sodium nitroprusside or glyceryl trinitrate
        - avoid beta blockers
- hyperthermia
    - benzodiazepines
    - aggressive management to prevent multi-organ failure
        - external cooling with ice packs and cool fluids
        - in conscious patients this is uncomfortable and causes shivering, so paralysis, intubation and ventilation should be considered

- ecstasy
    - background
        - contains mainly 3,4-methylenedioxy-N-methylamfetamine (MDMA)
        - class A drug
        - tablets may also contain other substances
        - may be tablets or capsules of different colours with different designs and pictures
            - can also be in powder or crystal form
        - has stimulant and mild hallucinogenic properties
            - effects can last for several hours

        - reported effects include initial rushing feeling followed by a feeling of energy, lack of aggression, empathy with others, greater appreciation of music and increased sexual and sensual experience
- adverse effects
    - immediate adverse psychological effects – more likely if the user is already anxious or takes a high dose
        - anxiety, panic, confusion, unpleasant distortion of the senses
        - effects can last for days or weeks
    - organic symptoms
        - tightening of the jaw
        - nausea
        - sweating
        - loss of appetite
        - dry throat and mouth
    - objective physiological signs
        - pupil dilation
        - hypertension
        - tachycardia
    - immediate post-drug period
        - extreme tiredness
        - sleep may last several days
    - psychological dependence can occur (physical dependence not known)
    - serotonin syndrome
        - more likely if other drugs taken that increase serotonin levels
- clinical features
    - sweating
    - dry mouth
    - anxiety
    - dehydration
    - hyponatraemia
    - hypertonia
    - hyperreflexia
    - hallucinations
    - hypertension
    - supraventricular arrhythmia
    - coma
    - convulsions
    - haemorrhagic CVA
    - rhabdomyolysis
    - metabolic acidosis
    - DIC
    - multi-organ failure
- management

        - benzodiazepines
        - cooling
        - possibly dantrolene – no evidence
        - avoid beta-blockers
- crystal methamphetamine
    - background
        - amphetamine-type stimulant
        - blocks presynaptic catecholamine reuptake within the CNS, causing hyperstimulation at the post-synapse
        - most commonly smoked in crystal form in a pipe or aluminium foil, heated by a flame
            - can also be snorted, injected, swallowed in pill form or inserted rectally
        - commonly administered in runs of several days
            - individuals may stay awake for up to 10 days with little food or drink and prolonged sleep or mild dysphoria as the drug wears off
        - use is uncommon in the UK
    - presentation
        - euphoria
        - increased alertness
        - increased energy
        - increased libido and enhanced sexual pleasure
        - decreased inhibition
        - decreased appetite
        - decreased need for sleep
    - physiology
        - peripheral (indirect sympathomimetic effects)
            - tachycardia
            - hypertension
            - palpitations
            - tachypnoea
            - sweating and hyperthermia
            - dry mouth
            - decreased GI motility
            - dilated pupils
            - tremor
        - central
            - jaw clenching
            - repetitive jerking and choreoathetoid movements
            - compulsion with repetitive tasks (‘punding’)
            - formication (sense of flesh crawling with bugs with associated picking and sores)
    - common presentations to healthcare
        - diarrhoea and nausea
        - insomnia
        - anxiety

- restlessness, agitation and irritability
- chest pain
    - myocardial infarction
    - dissecting aortic aneurysm
- panic attacks
- paranoia
- psychosis
- renal failure
- rhabdomyolysis
- seizures
- sexually and parenterally transmitted infections
- stroke
- trauma
- withdrawal symptoms:
    - depression
        - may be severe and prolonged and require monitoring for suicidal ideation
    - anxiety
    - irritability
    - problems concentrating
    - psychomotor slowing
    - increased appetite
    - paranoia
    - seizures may occur

- investigations may include:
    - bloods
        - FBC, U&E, CK, cardiac enzymes
        - ECG
        - CXR – if pulmonary symptoms
        - CT scan – if altered mental status
- management
    - acute intoxication
        - consider staff safety
            - sedation with neuroleptics (e.g. haloperidol) may be required
        - management is largely supportive
        - activated charcoal only helpful after oral ingestion
        - benzodiazepines for seizures
        - cooling measures if hyperthermia
        - hypertension may require IV beta-blockers
- complications
    - myocardial infarction
    - dissecting aortic aneurysm
    - cardiomyopathy
    - hypertensive crises and stroke
    - rhabdomyolysis and renal failure
    - erectile dysfunction

- GI ulcers and ischaemic colitis
- pulmonary hypertension
- skin abscesses and lesions
- premature ageing
- cognitive impairment
    - confusion, memory loss, motor slowing, learning impairment
- dental decay ('meth mouth')
- psychiatric disorders
    - anxiety
    - depression
    - psychosis
    - suicidal ideation and behaviour
- malnutrition and weight loss
- dependence and withdrawal-related symptoms
- trauma
- in pregnancy:
    - placental insufficiency and abruption
    - intrauterine growth restriction
    - prematurity
    - cleft palate defects
    - cardiac anomalies
    - miscarriage and stillbirth

- opioids
    - clinical features
        - miosis
        - CNS depression
        - respiratory depression
        - complications of hypoxia:
            - seizures
            - dysrhythmia
            - brain injury
    - management
        - resuscitation
        - electrolytes and acid-base
            - respiratory acidosis
        - specific therapy
            - naloxone in titrated doses until reversal of respiratory depression
                - may require infusion
            - dose 0.4-2mg IV/IM/SC or 1-10μg/kg/h

PhC2 poisoning – carbon monoxide, cyanide, organophosphate

- carbon monoxide poisoning
    - colourless, odourless gas produced by incomplete combustion of a hydrocarbon or carbohydrate
        - e.g. gas, coal, petrol, diesel, paraffin or wood, paper

- approximately 50 accidental deaths/year, likely more
- pathophysiology
    - carbon monoxide binds to haemoglobin 240 times more strongly than oxygen
    - shifts oxyhaemoglobin dissociation curve to left
    - presence of CO on one of the four oxygen molecule binding sites on haemoglobin causes oxygen to bind with greater affinity
        - so less readily released to tissues
        - can be hypoxic with a normal PaO2
    - CO also inhibits cytochrome oxidase, preventing cells from using the reduced amounts of oxygen they receive
    - may also increase adhesion of inflammatory cells and platelets to the capillary endothelium, with leakage of fluid across cerebral capillaries causing cerebral oedema
        - unconscious patients may have delayed damage due to leuko-encephalopathy
            - damage usually at 'watershed' areas such as basal ganglia
    - half life of CO is:
        - 5-6 on room air (21%)
        - 1.5 hours on 100% oxygen
        - 23 minutes on hyperbaric oxygen (2 atmospheres)
- clinical features:
    - headache, nausea, vomiting, malaise
    - neurological: ataxia, lack of co-ordination, agitation, impaired conscious level, retinal haemorrhage, hyperreflexia, weakness, extensor plantars, convulsions
    - respiratory: hyperventilation, pulmonary oedema, respiratory failure
    - cardiac: chest pain, MI, arrhythmia
    - dermatological: cherry red skin (rare), blisters
    - chronic symptoms are very non-specific (headache, nausea, flu-like symptoms); patients may later develop neuropsychiatric features (memory impairment, disorientation, apathy, personality change, parkinsonism, gait disturbance)
        - consider if:
            - more than one person affected in the house
            - symptoms better when away from the house
            - symptoms related to cooking (when stove in use)
            - symptoms worse in winter (when heating in use)
- severity indicators:
    - any new objective neurological sign (e.g. increased tone, upgoing plantars)
    - coma
    - need for ventilation
    - ECG indication of infarction or ischaemia
    - clinically significant acidosis
    - initial carboxyhaemoglobin level >30% (weak link with clinical outcome)
- investigations:
    - COHb – breath analyser or blood test

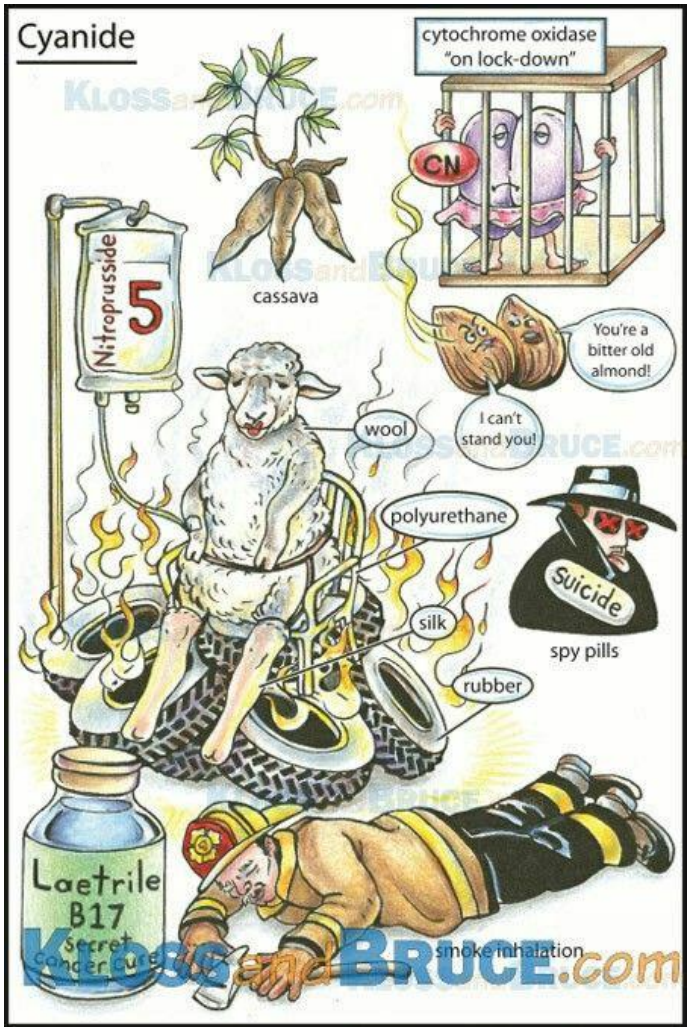
                - level depends on time since exposure, smoking status, percentage of oxygen breathed
            - oxygen saturations
                - read falsely high due to similar light absorbency of carboxyhaemoglobin and oxyhaemoglobin
                - should not be used to guide need for supplemental O2
            - ECG
            - ABG (evidence of metabolic acidosis)
            - CK (risk of rhabdomyolysis)
            - renal function
        - management:
            - high flow oxygen with tight fitting face mask
            - supportive fluid resuscitation
            - consider sodium bicarbonate if metabolic acidosis persists despite oxygen and fluid resuscitation
            - mannitol if cererbral oedema suspected (1g/kg over 20 minutes)
            - appliances and flues need to be checked before patient goes home
                - contact social services if needed
            - hyperbaric therapy not recommended as insufficient evidence for long distance transfers
                - indications if available locally are: loss of consciousness at any stage, neurological signs other than headache, myocardial ischaemia or arrhythmia, pregnancy, COHb >20%
- cyanide poisoning
    - can be in liquid or gas form
    - sources include:
        - smoke inhalation
            - burning plastics, wool, silk, natural and synthetic polymers
        - cyanogenic glycosides such as amygdalin
            - almonds, apricot kernels, other Prunus species such as peach, apple, cherry, plum
        - sodium nitroprusside
        - industrial exposure
            - cyanide salts for metal extraction and refining, electroplating, photography, fumigation
        - acetonitrile
            - industrial solvent used as cosmetic remover and in laboratories
        - chemical warfare and acts of terrorism
        - poison for animal control
            - e.g. rodenticide
        - alternative medicines
            - e.g. derived from apricot kernels
        - fumigant in aeroplanes, buildings, ships
    - pathophysiology:
        - binds ferric (Fe3+) ion of cytochrome oxidase, causing hypoxia and lactic acidosis

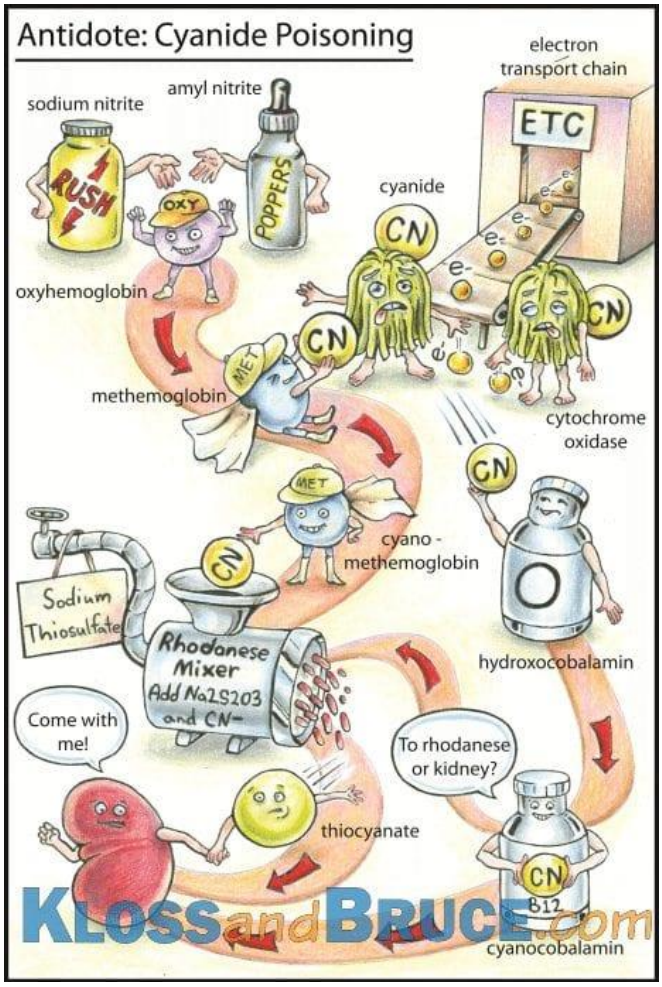
        - stimulates biogenic amine release causing pulmonary oedema and heart failure due to vasoconstriction
        - stimulates neurotransmitter release, causing neurotoxicity and seizures
        - precursors:
            - acetonitrile is metabolised by liver and can lead to toxicity over 24 hours
            - sodium nitroprusside is metabolised to cyanide and can accumulate with prolonged high dose infusions
    - clinical features:
        - acute inhalation or ingestion
            - rapid loss of consciousness and seizures with inhalation
            - onset of symptoms over around 30 minutes with ingestion
        - milder exposures
            - non-specific symptoms such as nausea, vomiting, headache, dyspnoea, increased respiratory rate, hypertension, tachycardia, altered conscious level, seizures
        - severe exposures
            - progressive features from end-organ damage
            - hypotension, bradycardia, reduced GCS, respiratory depression, cardiovascular collapse
            - hyperlactaemia
            - may appear pink due to high SvO2 after oxygen administration
            - may have odour of bitter almonds (not detectable by everyone)
    - investigations:
        - bloods gas
            - lactate >10
            - high SvO2 with oxygen administration (poor oxygen extraction)
            - COHb (suspect co-existent CO poisoning if smoke inhalation
        - cyanide levels if available
    - management:
        - removal from source
        - PPE
            - danger to healthcare workers from dermal route and inhalation
            - patient vomit can liberate hydrogen cyanide gas
        - resuscitation
            - high flow oxygen
            - haemodynamic support (may need inotropes/vassopressors)
            - antidote (ideally hydroxocobalamin then sodium thiosulfate)
        - supportive care
        - address suicidality if indicated
        - address burns
        - asymptomatic patients with normal blood gases can be discharged at 6 hours
    - antidotes
        - are cobalt-containing, sulphur donors or methaemoglobin generators
        - hydroxocobalamin and thiosulphate:
            - less toxic than dicobalt edetate

- 5g hydroxocobalamin in 200ml 5% dextrose over 30 minutes, then 12.5g sodium thiosulphate (50ml of 25% solution) over 10 minutes
- repeat both if no improvement within 15 minutes
- adverse effects:
    - hydroxocobalamin – occasional hypertension, bradycardia or tachycardia, orange-red skin and body fluids for up to 48 hours
    - sodium thiosulphate – nausea and vomiting with rapid injection, hypotension, headache, abdominal pain, confusion

▪ dicobalt edetate
- 300mg over 1 minutes followed by 50ml 50% glucose
- repeated up to 3 times if immediate clinical response not seen
- salt is toxic – causes seizures, chest pain, dyspnoea, head and neck swelling, hypotension, urticaria, vomiting; only give if severe cyanide poisoning strongly suspected

▪ methaemoglobin generators (e.g. amyl nitrite – inhaled, sodium nitrite – IV)
- effectively contraindicated in smoke inhalation and possible CO poisoning as likely to aggravate tissue hypoxia





- organophosphates
    - pathophysiology:
        - cholinergic toxidrome
        - inhibition of acetylcholinesterase resulting in increased levels of acetylcholine
        - stimulation of muscarinic receptors results in excessive parasympathetic activity
        - stimulation of nicotinic receptors results in persistent muscle depolarisation
        - once the ‘ageing’ process occurs, AChE cannot be reactivated by pralidoxime
            - irreversible loss of an alkyl side chain causing irreversible permanent binding to AChE
    - causes include:
        - insecticides/fertilisers
        - surface and room sprays
        - bait for cockroaches
        - head lice shampoos
        - livestock dipping
        - fumigation
        - nerve agents (sarin)
    - features:
        - parasympathetic (DUMBBELS)
            - **d**iarrhoea

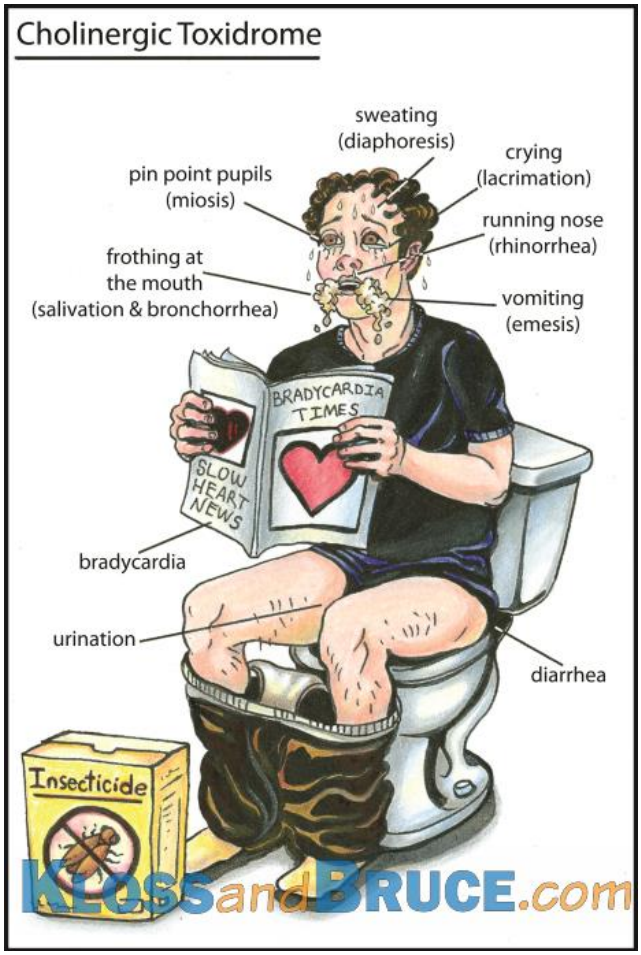
                - **u**rination
                - **m**iosis (small pupils)/**m**uscle weakness
                - **b**ronchorrhoea
                - **b**radycardia
                - **e**mesis
                - **l**acrimation
                - **s**weating/**s**alivation
            - nicotinic
                - weakness
                - fasciculations
                - paralysis
            - CNS
                - drowsiness
                - seizures
    - management:
        - decontamination
        - resuscitation
        - specific therapy:
            - atropine
                - 0.02mg/kg boluses
                - every five minutes, doubling dose each time
                - titrate until secretions dry up
                - treats bradycardia, hypotension, excess secretions
            - pralidoxime
                - 30mg/kg IV
                - then 8mg/kg/hr
                - treats muscle weakness
            - benzodiazepines
                - midazolam 0.2mg/kg
                - seizures or agitation
            - activated charcoal
                - consider if presenting within 1-2 hours

PhC3 toxidromes

- anticholinergic
    - causes
        - nicotinic or muscarinic receptor antagonism
        - antidepressants (e.g. tricyclics), antipsychotics, antihistamines
    - features
        - ‘blind as a bat, mad as a hatter, red as a beet, hot as hell, dry as a bone’
        - dry skin
        - dry mouth
        - thirst
        - hyperthermia
        - dilated pupils (mydriasis)
        - tachycardia
        - urinary retention

        - decreased bowel sounds

- cholinergic
    - causes
        - overstimulation of nicotinic/muscarinic receptors
        - organophosphates
    - features
        - defecation
        - urination
        - small pupils (miosis)
        - bradycardia (muscarinic stimulation) or tachycardia (nicotinic stimulation)
        - emesis
        - lacrimation
        - sweating



- opioid
    - causes
        - opioids
    - features
        - CNS depression
        - hypoventilation
        - hypotension
        - pin-point pupils
        - pulmonary oedema
        - rapid response to appropriate dose of naloxone
- sympathomimetic
    - causes
        - cocaine, amphetamines
    - features
        - CNS excitation
        - seizures
        - tremor
        - hyperreflexia
        - hyper/hypotension
        - tachycardia
- serotonin
    - causes
        - overstimulation of 5HT2 receptors

        - triad of altered mental status, neuromuscular hyperactivity and autonomic instability
        - often two or more agents that act on serotonin syndrome
        - SSRIs, MAOIs, tricyclics, venlafaxine, MDMA (ecstasy), amphetamines, cocaine, tramadol, triptans, linezolid, St John's Wort
    - features
        - agitation
        - restlessness
        - confusion
        - clonus
        - tremor/shivering
        - hyperreflexia/hypertonicity/rigidity
        - fever/flushing

PhC4 use of antidotes

- immediate availability

What is it?	What is it for?	Notes
Acetylcysteine	Paracetamol	Prevents NAPQI accumulating when glutathione is depleted.
Activated charcoal	Many oral poisons	Don't use for PHAILS (**p**esticides, **h**eavy metals, **a**cids/alkalis/alcohols, **i**ron, **l**ithium, **s**olvents).
Atropine	Organophosphorus or carbomate insecticides; bradycardia	2mg every 5-10min for organophosphate poisoning (only treats muscarinic effects).
Calcium chloride	Calcium channel blockers Systemic effects of hydrofluoric acid	To reverse blockage of calcium channels in cardiac myocytes, vascular smooth muscle, pancreatic beta cells.
Calcium gluconate Calcium gluconate gel	Local infiltration for hydrofluoric acid Hydrofluoric acid	Do not give local anaesthetic, use pain to guide treatment. Can make gel with 10ml 10% calcium gluconate in 30ml lube if needed.
Cyanide antidotes: Hydroxocobalamin (Cyanokit) Sodium nitrite Sodium thiosulphate	Cyanide	Consider giving after smoke exposure with metabolic acidosis and lactate >10.
Flumazenil	Reversal of iatrogenic over-sedation with benzodiazepines	Generally safer to give supportive care and wait it out.
Glucagon	Beta-adrenoreceptor blocking drugs Other indications e.g. calcium channel blockers/TCA	Consider high dose insulin euglycaemic therapy instead (preferably via central line).

Intralipid 20%	Severe, systemic local anaesthetic toxicity	Also worth a try in arrest from propranolol, tricyclics, verapamil.
Methylthioninium chloride (methylene blue)	Methaemoglobinaemia	Treat if MetHb >30% or patient symptomatic/has cardiac risk factors. Sats will appear to go down initially. High doses can cause methaemoglobinaemia.
Naloxone	Opioids	Observe patient for at least 6 hours afterwards. Start infusion at 60% of dose needed to get good response for 15 minutes.
Procyclidine injection	Dystonic reactions	Only works for secondary causes (i.e. drugs). Can discharge with 3 day oral course.
Sodium bicarbonate 8.4% and 1.26%	TCAs and class Ia and Ic antiarrhythmic drugs Urinary alkalisation	Use for arrhythmias or long QT. Manage severe metabolic acidosis with hyperventilation and improved perfusion in ventilated patients.
ViperaTAb	European adder, Vipera berus	For shock/angioedema/acidosis, rapid local swelling, significant swelling within 48 hours. Remember antivenom can cause anaphylactoid reaction.

- available within 1 hour

Calcium folinate	Methotrexate Methanol, formic acid	Features include vomiting, diarrhoea, mucositis, rash, bone marrow suppression and can be precipitated by AKI.
Cyproheptadine	Serotonin syndrome	Triad of features of serotonin syndrome: CNS dysfunction, autonomic disturbance, neuromuscular effects. Cyproheptadine is an antihistamine with antiserotinergic effects.
Dantrolene	Neuroleptic malignant syndrome Other drug-related hyperpyrexia	Dantrolene is a muscle relaxant which prevents calcium release at the sarcoplasm. Can cause irreversible fatal hepatotoxicity and tissue necrosis (on extravasation).
Desferrioxamine	Iron	Requires cardiac monitoring.
Digoxin specific antibody fragments (Digifab)	Digoxin and related glycosides	Acute effects of digoxin toxicity include nausea/vomiting, abdo pain, bradycardia and/or ectopics, bigeminy, SVT, VT, hypotension, lethargy, confusion.
Fomepizole (or, if unavailable, ethanol IV or PO)	Ethylene glycol, diethylene glycol, methanol	A mouthful of ethylene glycol can be fatal for a child. Consider: history of ingestion, osmolar gap >10

		mOsm/L, acidosis or bicarb <20, anion gap >11, urinary oxalate crystals
Idarucizumab	Dabigatran related active bleeding (discuss with local haematologists and NPIS)	Monoclonal antibody fragment, works immediately, lasts for 12 hours.
Macrogol ’3350’ (polyethylene glycol) Klean-Prep	Whole bowel irrigation for agents not bound by activated charcoal (e.g. iron, lithium), also for bodypackers and for slow-release preparations	Rarely used – lack of evidence base, risks to patient (including airway risk), labour-intensive (cannot leave patient). If done, aim for clear effluent.
Mesna (in hospitals commonly using cyclophosphamide)	Cyclophosphamide	Reduces risk of urotoxicity, rarely if ever going to be used in the ED.
Octreotide	Sulphonylureas	Somostatin analogue. One sulphonylurea tablet can be fatal in a child and cause significant hypoglycaemia in an adult, can last for days. Correct with 50ml 50% glucose first, then give octreotide.
Phentolamine	Digital ischaemia related to injection of epinephrine	Alpha blocker. Can be used if there is inadequate perfusion in the digit, mixed with lidocaine and infiltrated into the puncture site.
Phytomenadione (Vitamin K1)	Vitamin K dependent anticoagulants	Takes 6-12 hours to start working when given IV. Give PCC if active bleeding/haemodynamic instability.
Protamine sulphate	Heparin	Rarely required as stopping the heparin infusion is usually sufficient. Only partial reversal for LMWH. Can cause hypotension and anaphylaxis (particularly in people with fish allergy or vasectomy)
Pyridoxine, high dose injection	Isoniazid	Other sources of hydrazine include rocket fuels, manufacture of insecticides, dyes, plastics, certain mushrooms.

- available supraregionally

Prussian blue (Berlin blue)	Thallium	Thallium is used in jewellery, glass and semiconductor manufacturing and is odourless and tasteless.
Botulinum antitoxin	Botulism	Botulinum toxin prevents release of acetylcholine at neuromuscular junction.

		Usually from canned food, can be from injections/wound infection.
Glucarpidase	Methotrexate	Used when there is a significant deterioration in renal function and toxic plasma methotrexate levels.
Pralidoxime chloride	Organophosphurus insecticides	Reactivated acetylcholinesterase by removing the phosphate group if given in 1-2 hours before bond becomes irreversible. Treats the cholinergic effects only.
Sodium calcium edetate	Heavy metals (particularly lead)	Increases renal excretion of lead, should not be used if patient anuric.
Succimer (DMSA)	Heavy metals (particularly lead and arsenic)	Most heavy metals cause GI symptoms then multi-organ failure. Consider whole bowel irrigation if radio-opaque matter on x-ray.
Unithiol (DMPS)	Heavy metals (particularly mercury)	More toxic than succimer but can be given IV.

PhC5 batteries, household chemicals, poisonous plants

- batteries
    - button batteries
        - generate hydroxide ions at negative pole (the narrowest side on the lateral x-ray) producing localised alkaline corrosive injury with tissue liquefaction and necrosis
            - can develop within 2 hours
            - severity related to size of battery, current produced, length of time
        - complications:
            - oesophageal perforation
            - trachea-oesophageal fistula
            - aorto-oesophageal fistula
            - stricture
        - risk assessment:
            - battery size
                - >20mm can cause severe local damage within 2 hours
                - <20mm less likely to lodge in oesophagus
                - smaller batteries can cause localised damage in aural or nasal cavities
            - age
                - most fatalities are in under 4s
                - consider non-mobile infants fed by siblings and elderly patients mistaking batteries for tablets
            - time
                - delayed diagnosis has worse outcome
                - batteries can still produce charge for 10 years, ‘spent’ batteries can still cause damage
                - perforation and fistulas may not be evident for 28 days
        - clinical features:

- normally asymptomatic
    - shiny object seen in mouth, batteries missing from device
- consider when:
    - airway obstruction, drooling
    - acute stridor or recurrent stridor
    - unexplained wheeze
    - cough, gagging, choking when eating and drinking
    - fever (indication of oesophageal perforation)
    - dysphagia/sore throat
    - chest discomfort
    - 'off food'
        - worth considering foreign body if no cause identified
    - vomiting
    - unexplained nasal, ear, rectal, vaginal or eye discharge/bleeding
    - haematemesis, melaena, haematochezia, epistaxis
        - late signs

- investigations:
    - plain AP x-ray of neck, chest, abdomen (or skull)
        - 'neck to bottom' x-rays for all GI bleeding and suspected foreign body ingestion or history or epistaxis with no bleeding point (may be GI)
        - do not mistake button battery for coin
            - 'double ring' appearance on PA (but absence does not exclude diagnosis)
            - 'step off' appearance on lateral
- management:
    - endoscopic removal of batteries in oesophagus within 2 hours of ingestion
    - urgent removal of batteries in ear or nose
    - risk of complication low if battery in stomach
        - repeat x-ray in 2 days if asymptomatic
            - discuss with upper GI surgeon if not moved then
    - if beyond pylorus and asymptomatic can be observed at home with repeat x-ray in 10-14 days (unless observed in stool prior to this)
    - discharge advice:
        - return if concerned, abdo pain, bloody vomit or faeces, problems swallowing, respiratory distress, persistent cough

- household chemicals
    - corrosives
        - examples include:
            - sodium hydroxide
                - detergents, drain and oven cleaners, button batteries
            - sodium hypochlorite
                - bleach, household cleaners

        - dilute solutions less than 150ml do not cause significant corrosive injury
    - ammonia
        - metal and jewellery cleaners
    - hydrochloric acid
        - metal cleaners
    - sulphuric acid
        - drain cleaners, car batteries
- mechanism:
    - direct chemical injury
        - depends on pH, concentration, volume ingested
        - acidic agents cause protein denaturation and coagulative necrosis alkaline agents are more dangerous, causing liquefactive necrosis with deep and progressive mucosal burns
    - some agents have systemic effects
- clinical features:
    - immediate injury to GI tract
        - mouth and throat pain
        - drooling
        - odynophagia
        - vomiting
        - abdominal pain
    - upper airway injury
        - progressive stridor
        - hoarseness
        - respiratory distress
- complications:
    - acute
        - oral, oesophageal, gastric burns (areas of anatomic narrowing most affected – cricopharyngeal, diaphragmatic oesophagus, antrum, pylorus)
        - oesophageal/gastric perforation
        - shock
        - haemorrhage
        - mediastinitis
        - organ failure
        - acidosis
        - external injury
    - chronic
        - laryngopharyngeal fibrosis with airway incompetence and chronic aspiration
        - oesophageal fibrosis, stricture, stenosis, pseudodiverticulum
        - gastric outlet obstruction
        - GORD
        - psychosocial problems

        - oesophageal carcinoma (1000 times increased risk, may occur up to 40 years later)
    - investigations:
        - chest and abdo x-ray if signs/symptoms of perforation
        - endoscopy within 24 hours
            - unless asymptomatic at 4 hours
            - endoscopic grades:
                - 0 – normal
                - I – mucosal oedema, hyperaemia
                - IIA– superficial ulcers, bleeding, exudates
                - IIB – deep focal or circumferential ulcers
                - IIIA – focal necrosis
                - IIIB – extensive necrosis
    - management:
        - immediate
            - manage airway compromise, GI perforation/sepsis
            - treat systemic toxicity
                - calcium for hypocalcaemia
                - methylene blue for methaemoglobinaemia
                - avoid excess O2 for paraquat
        - supportive
            - keep NBM if symptomatic
            - do not insert NG tube pre-endoscope
            - antibiotics if evidence of perforation
        - decontamination
            - rinse mouth with water
            - do not induce vomiting, administer oral fluids or activated charcoal, attempt pH neutralisation or perform gastric lavage
    - disposition:
        - if asymptomatic at 4 hours, trial of oral fluids and home if tolerated
        - consider endoscopy even for asymptomatic patients
        - if symptomatic (throat pain, drooling, pain on swallowing own saliva, vomiting, abdominal pain) – NBM and observe, endoscopy within 24h
        - airway compromise – ITU
        - perforation/sepsis/instability – surgical assessment and ITU
- poisonous plants
    - rare to have severe toxicity in humans
    - identification and toxin quantification can be difficult (variation with species, plant part, life cycle, season, location)
    - exposure:
        - usually young children or mistaking for edible variety
        - recreational use, alternative medicines, self-harm
        - any part of plant (root, leaves, berries, seeds), may be raw, cooked, infused
        - cutaneous or ocular exposure may be symptomatic
    - examples:

- aconite (e.g. Acontium and Delphinum spp)
    - may be in Asian herbal medicines
    - sodium channel activator
    - GI: nausea and vomiting, abdominal cramps, diarrhoea
    - CVS: tachycardia, dysrhythmia, shock
    - CNS: paraesthesia, paralysis, coma, seizures
    - multi-organ dysfunction, lactic acidosis
- belladonna alkaloids (e.g. Datura spp, Atropa belladonna, Hyoscyamus niger – henbane)
    - anticholinergic syndrome
- calcium oxalate crystals (e.g. Dieffenbachia spp, Philndendron spp)
    - severe mechanical irritation of mucous membranes
- cardiac glycosides (e.g. Digitalis purpurea – foxglove, Nerium spp – pink oleander, Thevetia spp – yellow oleander)
    - mimics digoxin toxicity
    - GI symptoms, cardiotoxicity (AV blockade, increased automaticity, dysrhythmia)
- colchicine (e.g. Colchicum autumnale – autumn crocus, Gloriosa superba – glory lily)
    - GI symptoms
    - bone marrow failures
    - multi-organ failure
- coniine (e.g. Conium maculatum – poison hemlock)
    - alkaloid similar to nicotine
    - GI symptoms, dysrhythmia, ascending paralysis, rhabdomyolysis, renal failure
- cyanogenic glycosides such as amygdalin (e.g. Prunus seed kernels – apricot, plum, pear, cherry)
    - hydrolysed to form cyanide
    - coma, lactic acidosis, multi-organ failure, shock
- hypoglycin (e.g. Blighia sapia – ackee)
    - hypoglycaemia, acidaemia, vomiting, coma, seizures
- nicotine (e.g. Nicotiana spp – tobacco)
    - ingestion, inhalation or transdermal exposure
    - nicotinic syndrome – GI symptoms, sweating, tachycardia, hypotension, tremor, seizures
- psychotropic alkaloids (e.g. Ipomea spp – morning glory, Lophophora wiliamsoni – peyote cactus)
    - e.g. direct serotonin agonists like lysergic acid (LSD) and mescaline
    - psychosis including visual hallucinations
- ricin (e.g. Ricinus communis – castor beans) and abrin (e.g. Abrus precatorius – jequirity beans)
    - similar antimitotic effects to colchicine
    - GI symptoms, bone marrow failure, multi-organ dysfunction
- taxine (e.g. Taxus spp – yew)
    - sodium and calcium channel inhibition
    - GI symptoms

                - bradycardia
                - dysrhythmias
                - altered mental state
        - investigations
            - guided by clinical assessment
            - digoxin levels do not correlate with severity from plant cardiac glycosides
            - consider FBC, U&E, LFT, CK, glucose, ECG, lactate
        - management:
            - resuscitation
                - rarely needed
                - life-threatening features include:
                    - cardio-toxicity and shock
                    - seizures and coma
                    - multi-organ failure
                    - hypoglycaemia
                    - anaphylaxis
            - supportive care
                - neuro obs
                - delirium management
                - glucose monitoring
                - cardiac monitoring
                - rehydration, antiemetics
                - treatment of dermatitis
            - decontamination
                - active charcoal if potential for severe toxicity
                - irrigation of affected eyes, mucous membranes, skin
            - antidotes
                - anticholinergic syndrome – physostigmine
                - cyanogenic glycosides – hydroxocobalamin and sodium thiosulfate
                - cardiac glycosides – digoxin immune Fab
                - colchicine – antidote available in France!

RESPIRATORY

RP1 chest pain

- potentially life-threatening causes of chest pain
    - cardiovascular
        - acute coronary syndromes
        - aortic dissection
        - myocarditis
            - most common cause of sudden death in the young
        - pericarditis
        - pulmonary embolism
    - other

        - tension pneumothorax
        - acute chest syndrome (sickle cell disease)
        - Boerhaave syndrome
- common non-life threatening causes of chest pain
    - gastrointestinal
        - biliary colic
        - gastroesophageal reflux
        - peptic ulcer disease
    - pulmonary
        - pneumonia
        - pleurisy
    - chest wall syndromes
        - musculoskeletal pain
        - costochondritis
        - thoracic radiculopathy
        - Bornholm disease
            - acute transient viral myositis associated with Coxsackie B)
        - Tietze syndrome
            - idiopathic benign inflammation of one or more of the costal cartilages
        - Texidor twinge
            - precordial catch syndrome
        - Mondor's disease
            - thrombophlebitis of the superficial veins of the breast and chest wall
    - psychiatric
        - anxiety
        - somatisation
        - Da Costa syndrome
            - symptom-complex characterised by palpitations, dyspnoea, precordial pain, fatigue, exaggerated emotional responses with increased cardiac awareness
    - occult trauma
        - rib fractures
    - infection
        - shingles

RP2 breathlessness

- terms
    - dyspnoea
        - overall term to describe an unpleasant awareness of respiratory effort
    - tachypnoea
        - respiratory rate greater than normal
    - hyperpnoea
        - greater than normal level of ventilation, which may be normal to meet metabolic requirements (e.g. exercise)
    - hyperventilation
        - overbreathing results in a lowering of alveolar and arteriolar pCO2

        - minute ventilation exceeds metabolic demand
    - orthopnoea
        - breathlessness on lying flat
        - due to redistribution of blood leading to an increased central and pulmonary blood volume and to abdominal contents pressing against the diaphragm in a recumbent position
    - paroxysmal nocturnal dyspnoea
        - sudden onset of dyspnoea usually occurring at night and waking the patient from sleep
        - mechanism is similar to orthopnoea but the sensory awareness of the developing situation is depressed by sleep
    - bradypnoea
        - inappropriately reduced respiratory rate which can occur when a patient becomes exhausted following prolonged tachypnoea or ingestion of certain toxins
    - Cheyne-Stokes breathing
        - abnormal breathing pattern characterised by progressively deeper and/or faster breathing, followed by a gradual decrease in depth/rate that results in temporary apnoea and is caused by damage to the respiratory centres in the brain
- pathophysiology
    - poorly understood
    - normal breathing is controlled by respiratory centres in the brainstem
    - the vagus nerve carries afferent and efferent components and provides the pathway for the neurological input resulting in the sensation of dyspnoea
    - other contributors to pathways leading to dyspnoea include:
        - intrapulmonary parenchymal stretch receptors
        - carotid body and central medullary chemoreceptors
        - peripheral vascular receptors
        - pulmonary artery baroreceptors
    - 85% of cases of dyspnoea are accounted for by asthma, cardiac failure, COPD, pneumonia, interstitial lung disease and psychogenic disorder
    - chemoreceptors
        - hypercapnia is well recognised as a cause of dyspnoea
        - the relationship is not straightforward and is mediated by changes in pH which can be compensated
        - there is uncertainty about the role of hypoxia as a cause of dyspnoea
    - mechanoreceptors
        - pulmonary stretch receptors are stimulated during lung inflation and provide feedback to terminate inspiration
        - there are also mechanoreceptors in the chest wall
- causes
    - pulmonary
        - airway obstruction
        - anaphylaxis
        - pulmonary embolism
        - spontaneous pneumothorax
        - asthma

        - COPD
        - pneumonia/pneumonitis/aspiration
        - pleural effusion
        - neoplasia
        - fibrosis
    - cardiovascular
        - pulmonary oedema
        - acute coronary syndrome
        - arrhythmia
        - pericardial tamponade
        - pericarditis/myocarditis
        - cardiomyopathy
        - valvular heart disease
        - congenital heart disease
    - infectious
        - epiglottitis
        - pneumonia
    - traumatic
        - pneumothorax
        - haemothorax
        - pericardial tamponade
        - flail chest
        - pulmonary contusion
        - chest wall injury
    - abdominal
        - ascites
        - obesity
        - pregnancy
    - psychogenic
        - anxiety/panic attack
        - hyperventilation
    - metabolic
        - metabolic acidosis
        - poisoning/toxin ingestion
        - renal failure
    - haematological
        - carbon monoxide poisoning
        - anaemia
    - neuromuscular
        - cerebrovascular accident
        - Guillain-Barré syndrome
- history
    - onset of dyspnoea
        - abrupt onset may suggest PE, anaphylaxis, pneumothorax
    - duration of symptoms
    - severity of dyspnoea
        - at rest or on exercise
            - what the exercise tolerance is

- precipitating events
  - trauma, allergen exposure, contact with infectious person etc.
- associated symptoms
  - chest pain
  - sputum
  - haemoptysis
  - fever
  - polydipsia and polyuria
  - anxiety or stress
- postural changes
- past medical history and previous episodes
- occupational and social history

- examination
  - respiratory
    - tachypnoea
      - non-specific indicator of respiratory distress
    - bradypnoea
      - may indicate impending respiratory arrest
    - abnormal respiratory pattern
      - may suggest intracranial event
    - stridor
      - upper airways obstruction
    - asymmetrical chest wall movement
      - pneumothorax, rib fractures
    - subcutaneous emphysema
      - pneumothorax, rib fractures
    - unilateral decreased air entry
      - pneumothorax, pleural effusion, collapse, consolidation
    - crepitations
      - inspiratory, fine – pulmonary oedema or fibrosis
      - expiratory, coarse – pneumonia
    - wheeze
      - asthma, COPD, focal obstruction
    - bronchial breath sounds
      - consolidation, pneumonia
    - friction rub
      - pleurisy, pulmonary embolus
  - cardiovascular
    - tachycardia
    - hypotension
      - PE, tension pneumothorax, sepsis, cardiac tamponade
    - elevated JVP
      - tension pneumothorax, cardiac tamponade, PE, pulmonary hypertension, CCF
    - cardiac murmur
      - valvular abnormality
    - added heart sounds

                - cardiac failure, massive PE
            - quiet heart sounds
                - cardiac tamponade
    - general
        - pyrexia
            - infection
        - weight loss
            - malignancy
        - clubbing
            - chronic respiratory conditions
            - congenital heart disease
        - pallor
            - anaemia
        - cyanosis
            - severe respiratory compromise, cyanotic heart disease
        - muscle wasting
            - neuromuscular disease
        - peripheral oedema
            - congestive cardiac failure
        - calf tenderness
            - PE
        - hand tremor/flap
            - CO_2 retention
- investigations
    - pulse oximetry
        - determine need for and response to oxygen supplementation
    - arterial blood gas
        - accurate determination of acid-base status and A-a gradient, hypercarbia
    - CXR
        - to support clinical diagnosis, assess severity of pathology, exclude certain pathologies
    - ECG
        - evidence of ischaemia or other cardiac pathology, right ventricular strain in PE

RP3 haemoptysis

- background
    - haemoptysis is the symptom of coughing up blood
    - massive haemoptysis is a life-threatening emergency with patients dying of asphyxia rather than blood loss, usually (95%) from the bronchial circulation under systemic pressure, more rarely (5%) from the pulmonary circulation
    - the most common causes of haemoptysis are chronic inflammatory conditions and lung malignancy
- causes
    - infection
        - mycobacteria, particularly TB
        - fungal infections

        - lung abscess
        - necrotising pneumonia (Klebsiella, Staphylococcus, Legionella)
    - iatrogenic
        - Swan-Ganz catheterisation
        - bronchoscopy
        - transbronchial biopsy
        - transtracheal aspirate
    - parasitic
        - hydatid cyst
        - paragonimiasis (lung fluke)
    - trauma
        - blunt/penetrating injury
        - suction ulcers
        - tracheoarterial fistula
    - neoplasia
        - bronchogenic carcinoma
        - bronchial adenoma
        - pulmonary metastases
        - sarcoma
    - children
        - bronchial adenoma
        - foreign body aspiration
        - vascular anomalies
    - vascular
        - pulmonary infarct
        - pulmonary embolism
        - mitral stenosis
        - arteriobronchial fistula
        - arteriovenous malformations
        - bronchial telangiectasia
        - left ventricular failure
    - coagulopathy
        - von Willebrand disease
        - haemophilia
        - anticoagulant therapy
        - thrombocytopaenia
        - platelet dysfunction
        - DIC
    - vasculitis
        - Behçet's disease
        - granulomatosis with polyangiitis
    - pulmonary
        - bronchiectasis (including cystic fibrosis)
        - chronic bronchitis
        - emphysematous bullae
    - miscellaneous
        - lymphangioleiomatosis
        - catamenial (endometriosis)

        - pneumoconiosis
        - broncholith
        - idiopathic
    - spurious
        - epistaxis
        - haematemesis
- investigations
    - bedside
        - ECG
        - echo
    - laboratory
        - FBC
        - coag
    - imaging
        - CXR
        - CT
        - echo
    - special tests
        - bronchoscopy
- management
    - goals
        - maintain airway patency
        - protect healthy lung
        - treat cause
        - fluid resuscitation
    - resuscitation
        - bleeding lung side down
        - if both sides bleeding, place head down
        - isolate lung
            - railroad ETT into non-bleeding lung with scope
    - specific therapy
        - treat cause
            - antibiotics
            - steroids
        - correct coagulopathy
        - bronchial artery embolization
        - bronchoscopic laser photocoagulation
        - iced normal saline lavage of involved lung segments
        - topical adrenaline
        - IV vasopressin
        - surgery

RP4 cough

- chronic cough
    - background
        - cough lasting more than 8 weeks
        - reported by 10-20% of adults

        - most common causes are smoking, postnasal drip, asthma and GORD
        - no cause is found for a substantial proportion of subacute and chronic cough
- physiology
    - the cough reflex is triggered by mechanical or inflammatory changes or irritants in the airway
    - the afferent pathway is via the vagus nerve to respiratory neurons termed the 'cough centre' in the brain stem
        - higher cortical centres also control the cough
    - chronic cough tends to be inhibited during sleep
    - chronic cough is often associated with bronchial hyper-reactivity, which can persist in the absence of the initiating cough event
        - defined as a state of increased sensitivity to a wide range or airway-narrowing stimuli (e.g. exercise, dry or cold air, hypertonic or hypotonic aerosols
        - occurs in asthma and COPD but can occur in the absence of disease
- common causes
    - smoking
        - active or passive
    - asthma
        - history of atopy
        - nocturnal cough
        - wheeze
        - peak flow rate variable by >20% or reversible changes on spirometry
            - absence of these does not rule out asthma
    - COPD
    - GORD
        - heartburn
            - may not have any GI symptoms
            - cough worse at night
            - cough when eating/talking
            - hoarseness
            - sour taste
    - postnasal drip
        - subjective symptoms – postnasal drip, having a recurrent need to clear the throat
        - persistent nasal blockage
        - persistent nasal discharge
    - environmental pollution (especially particles of 10 micrometres or fewer)
    - ACEi
    - occupational exposure to irritants
    - whooping cough
        - may be more common than previously supposed
- less common causes
    - cardiovascular
        - left ventricular failure
        - pulmonary emboli
        - aortic aneurysm

    - chronic infections
        - bronchiectasis
        - tuberculosis
        - cystic fibrosis
        - lung abscess
    - postinfectious cough
        - more common after Mycoplasma pneumoniae, chlamydial pneumonia, whooping cough
    - parenchymal lung diseases
        - interstitial lung fibrosis
        - emphysema
        - sarcoidosis
    - tumours
        - lung cancer
        - metastatic carcinoma
        - lymphoma
        - mediastinal tumours
        - benign tumours
    - upper airway conditions
        - chronic tonsil enlargement
        - obstructive sleep apnoea
        - chronic snoring
        - irritation of external auditory meatus
    - foreign body in large airways
        - recurrent aspiration
        - inhaled foreign body
        - endobronchial sutures
    - cardiac arrhythmia (rare)
    - cough only when supine
        - collapse of large airways
    - presentation of an involuntary tic
    - possible contribution of B12 deficiency (possibly due to sensory neuropathy)
    - idiopathic or psychogenic
- history
    - nature of cough
        - dry
        - sputum
        - blood
    - pattern of cough
        - duration
        - frequency
        - nocturnal
        - association with eating or talking
    - history of atopy
    - smoking status
    - occupation
    - drugs (especially ACEi)
    - red flags

        - copious sputum production
            - bronchiectasis
        - systemic symptoms (fever, sweats, weight loss)
            - TB
            - lymphoma
            - bronchial carcinoma
        - haemoptysis
            - tuberculosis
            - bronchial carcinoma
        - significant dyspnoea
            - heart failure
            - COPD
            - fibrotic lung disease
- examination
    - systemic signs
        - fever
        - weight loss
        - clubbing
        - lymphadenopathy
    - upper airway signs
        - hoarseness
        - nasal speech
    - focal chest signs
    - cardiovascular system
    - peak expiratory flow rate
- investigation
    - CXR
    - PEFR
    - bloods
        - FBC (infection, eosinophilia)
        - CRP (infection, malignancy, connective tissue disorders)
    - other tests as indicated by history
        - e.g. sputum analysis, bronchoscopy, echo, oesophageal pH testing or manometry, CT/MRI
- management
    - trial of treatment if appropriate (e.g. salbutamol for asthma, high dose PPI for GORD, antihistamines or nasal steroids for postnasal drip)
    - smoking cessation
    - treatment of underlying causes
    - treatment of cough
        - no effective treatments controlling the cough response
        - cough suppressants may be useful, particularly if sleep is disturbed, but have a risk of sputum retention
        - codeine may be effective but can cause dependence
        - sedating antihistamines may be helpful at night
        - mucolytics or steam inhalation with postural drainage may be helpful if mucous viscosity is problematic

        - simple linctus can help a dry cough and is safe and inexpensive
        - expectorants have no evidence of effectiveness
        - centrally acting neuromodulators such as gabapentin and pregabalin may be useful for chronic refractory cough
            - speech therapy techniques may also be helpful

RC1 asthma

- background
    - asthma affects around 4 million adults in the UK
    - on average 3 people a day die in the UK from an asthma attack
- pathophysiology
    - characteristics of asthma
        - bronchial hypersensitivity
        - bronchoconstriction
        - bronchial inflammation
        - recurring symptoms of reversible airway obstruction that vary in severity
    - a large number of cells and mediators are implicated, including:
        - eosinophils
        - mast cells
        - leukotrienes
        - prostaglandins
        - T-lymphocytes
        - macrophages
        - adhesion molecules
    - increased mucous secretion and bronchial oedema also contribute to obstruction
    - reasons for cardiac arrest in asthmatics include:
        - mucous plugging from severe bronchospasm causing asphyxia
            - commonest modality of death in asthmatics
        - prolonged hypoxia precipitating cardiac arrythmia
        - breath stacking due to air entering the lungs but not being able to escape
            - raised intrathoracic pressure can cause pneumothoraces or decrease venous return to the heart, causing circulatory collapse
- risk factors
    - more commonly diagnosed in boys than girls, but more common in women than men
    - more common in urban areas and lower socio-economic groups
    - strong associations include:
        - family history of asthma (particularly maternal)
        - history of another atopic condition
    - exacerbating factors may also be causative:
        - air pollution
        - passive smoking
        - environmental allergen exposure (e.g. dust mites)
        - urbanisation
    - occupational asthma can be caused by exposure to specific chemicals
        - may account for up to 15% of adult onset asthma
- diagnosis in adults

- clinical diagnosis as no one test, sign or symptoms is specific enough to confirm diagnosis
- clinical features that increase the probability of adult asthma:
  - more than one of:
    - wheeze
    - cough
    - difficulty breathing
    - chest tightness
  - particularly if the symptoms:
    - are worse at night/early morning
    - occur in response to exercise, allergen exposure or cold air
    - are triggered by aspirin or beta blockers
    - occur in the absence of a cold
    - personal or family history of atopic disorder or asthma
    - widespread wheeze on chest auscultation
    - otherwise unexplained low FEV1 or PEFR
    - otherwise unexplained peripheral blood eosinophilia
- clinical features that lower the probability of adult asthma:
  - symptoms with colds only
  - chronic productive cough without wheeze or breathlessness
  - dizziness, lightheadedness or paraesthesia peripherally
  - voice disturbance
  - cardiac disease
  - significant smoking history (>20 pack years)
  - repeated normal chest examination when symptomatic
  - repeatedly normal PEFR or spirometry when symptomatic
- failure of symptoms to improve with treatment should prompt specialist referral

- investigations for patients with intermediate probability of asthma:
  - spirometry/PEFR
    - an FEV1/FVC <0.7 is strongly suggestive of asthma
    - spirometry has advantages over PEFR in the stable patient:
      - less effort dependent
      - normal ranges are more robust
      - airway obstruction can be clearly documented
    - PEFR is portable and useful for self-monitoring (particularly when occupational asthma is suspected) and in the acute setting
  - treatment trials and reversibility testing
    - 400ml improvement in FEV1 (or 60l/min increase in PEFR) in response to either of the following strongly suggests asthma:
      - 400mcg inhaled salbutamol
      - 6 week trial of steroid inhaler (beclomethasone 200mcg BD or equivalent)
  - tests that may be done by respiratory specialists (high sensitivity but moderate specificity for asthma):
    - eosinophil count in sputum
    - exhaled nitric oxide concentration (eosinophilic inflammation)
    - methacholine or histamine challenge (airway hyperresponsivity)

- differential diagnosis
    - a new asthma diagnosis cannot be made on one visit to the ED
    - conditions that may mimic asthma include:
        - COPD
        - inhaled foreign body
        - anaphylaxis
        - airway stenosis
        - bronchiectasis
        - sarcoidosis
        - lung cancer
        - obliterative bronchiolitis
        - congestive cardiac failure
        - pulmonary embolism
        - pulmonary fibrosis
        - hyperventilation syndrome
        - GORD
        - rhinitis
        - chronic cough syndrome
        - vocal cord dysfunction
- history
    - time of onset of symptoms
    - abrupt or gradual onset
    - pleuritic chest pain
    - fever
    - cough (and nature of cough)
    - current medication
    - compliance with medication
    - allergies
    - current/recent use of steroids
    - frequency of use of salbutamol
    - any treatment given pre-hospital
    - any use of aspirin, NSAIDs, beta-blockers
    - pregnancy
    - previous best PEFR (in last 2 years)
    - smoking status
- features associated with increased risk of near-fatal or fatal asthma (consider admission regardless of severity of current episode)
    - history of severe asthma
        - previous near-fatal asthma (intubation, ICU)
        - previous admission for asthma in past year
        - requiring maintenance therapy of 3 or more classes of asthma medication
        - heavy use of beta-2 agonist
        - repeated ED attendances with asthma
        - 'brittle' asthma – uncontrolled despite maximum maintenance treatment or recurrent sudden severe episodes when asthma well controlled
    - presence of adverse psychosocial factor
        - non-compliance with treatment
        - failure to attend appointments/little GP contact

        - frequent home visits
        - denial
        - self-discharge from hospital
        - psychiatric illness, self-harm, depression
        - extreme situational stress (income, employment, marital, legal)
        - alcohol or drug abuse
        - obesity
        - learning difficulties
        - social isolation
        - childhood abuse
- examination
    - thorough examination of the cardiorespiratory system
        - wheeze
        - tachypnoea
        - absence of pyrexia
        - absence of crepitations
        - tachypnoea
        - findings that would point to another trigger for sudden worsening or an alternative diagnosis (e.g. pneumonia, pneumothorax, pulmonary oedema, PE)
    - adverse clinical features that can identify some patients with severe asthma (not specific individually or together and their absence does not rule out severe attack):
        - severe breathlessness (including too breathless to complete sentences in one breath)
        - tachypnoea
        - tachycardia
        - silent chest
        - cyanosis
        - accessory muscle use
        - altered consciousness or collapse
- investigations
    - pulse oximetry
    - PEFR
    - possible additional investigations if needed:
        - ABG
            - only if sats <92% or other life-threatening features
        - blood tests
            - unlikely to be helpful for initial management as will not be availably fast enough
            - WCC may be elevated if patient on steroids or has intercurrent infection
            - hypokalaemia may be caused by beta agonist use
                - rarely clinically significant
                - can be corrected if marked or with ECG changes
            - serum theophylline level if indicated
        - CXR
            - not recommended for acute exacerbations unless:

        - suspected pneumothorax, pneumomediastinum, pneumonia
        - life-threatening features
        - failure to respond to standard treatments
        - patients requiring ventilation
    - ECG
        - if severe symptoms, irregular pulse or persistent tachycardia despite treatment
        - sinus tachycardia and evidence of right heart strain are common
        - may be signs of hypokalaemia (flat T wave, ST depression, long QT, U wave)
    - POCUS
        - presence of lung sliding can rule out pneumothorax
        - absence of lung sliding is not enough to rule out pneumothorax in critically unwell patients
        - presence of a lung point is 100% sensitive and specific for pneumothorax when seen, but may not be seen in large pneumothoraces
- risk stratification
    - the British Asthma guidelines categories are:
        - mild exacerbation
            - PEFR >75% best or predicted
            - no features of acute severe asthma
        - moderate acute asthma
            - PEFR 51-75% best or predicted
            - no features of acute severe asthma
        - acute severe asthma
            - PEFR <50% best or predicted
            - respiratory rate 25/min
            - heart rate 100/min
            - inability to complete sentences in one breath
            - no life-threatening features
        - life-threatening asthma
            - reduced conscious level
            - signs of exhaustion
            - hypotension
            - arrhythmia
            - cyanosis
            - poor air entry/poor respiratory effort
            - silent chest
            - PEFR <33% best or predicted
            - SpO2 <92% or PaCO2 normal (4.6-6.0 kPa)
        - near fatal asthma
            - raised PaCO2 and/or needing high inflation pressures if ventilated
    - the PRF must be consulted to establish what the patient was like at initial presentation before any treatment
    - proformas in the ED have been shown to improve management

- management
    - oxygen
        - for any patient with sats <94% or if pulse oximetry not immediately available
        - titrate to maintain saturations between 94% and 98%
        - nebulisers should be driven by oxygen (at least 6 l/min)
    - beta 2 agonists
        - metered dose inhalers with spacers
            - 4 puffs of salbutamol then 2 puffs every 2 minutes up to a maximum of 10 puffs
            - suitable for patients with moderate exacerbations
        - nebulisers
            - for patients with severe exacerbations/needing oxygen
            - repeat doses of 5mg nebulisers can be given
            - in practice more than 10mg/hour is unlikely to improve effectiveness but may increase side effects
        - IV salbutamol
            - not shown to be superior to nebulisers but may be useful if they cannot be used effectively (patient pulling off mask/poor air entry)
    - ipratropium bromide
        - may not be beneficial for mild exacerbations
        - only for use in severe or life-threatening cases or cases not responding to salbutamol
        - side effects are uncommon but include dry mouth, dry eyes, blurred vision, tachycardia, flushing, confusion, urinary retention
        - long acting and should not be given more frequently than four hourly
            - if given more frequently, risk of side effects increases, particularly in the elderly
            - should not be given in the first 4 hours in the ED if it was given pre-hospital
    - steroids
        - for life-threatening asthma
            - PEFR <75% after initial salbutamol treatment; any indicator of severe asthma after initial therapy
        - consider two 25mg tablets or soluble prednisolone rather than 8-10 5mg tablets if patient at risk of hypoxia when oxygen removed
            - IV hydrocortisone 100mg QDS can be used if patient unable to swallow or vomiting but does not work faster
        - patients whose PEFR was initially <50% best or predicted who are being discharged should have a course of oral steroids
            - can be stopped after 5 days if the patient is continuing with inhaled steroids
            - longer courses may be required if the patient remains symptomatic or is on long term steroid maintenance therapy
    - magnesium sulphate
        - there is limited evidence of effectiveness or safety

        - guidelines suggest giving a single dose of IV magnesium to patients with acute severe asthma (PEFR <50% best or predicted) who have not had a good initial response to inhaled bronchodilators
        - 1.2-2g over 20 minutes
            - most common dose is 2g in 100ml 0.9% sodium chloride
        - risk of hypermagnesaemia with muscle weakness and respiratory fatigue
    - IV aminophylline
        - unproven benefit in life-threatening asthma and is arrhythmogenic
        - if it is going to be started, a level should be checked first for patients on aminophylline or theophylline
        - loading dose for those not on long term therapy is 5mg/kg over 20 minutes
        - maintenance dose is 0.5-0.7mg/kg/hr
    - IV fluid
        - no evidence to suggest that this improves outcomes
        - of use if patient is dehydrated or there is electrolyte imbalance
    - antibiotics
        - not recommended routinely – most triggers are viral
    - leukotriene antagonists
        - no evidence to support in acute exacerbations
- ongoing monitoring/investigations in the ED
    - heart rate, respiratory rate, BP and PEFR should be recorded 15-30 minutes after starting treatment and serially according to response
    - patients that stabilise should be assessed at one hour and two hours after initial treatment
    - oxygen saturations should be maintained at 94-98%
    - urgent speciality review for patients failing to improve or deteriorating after initial treatment
    - review potassium levels when U&Es available
    - if ABG was indicated, this should be repeated 1 hour after starting treatment
- British asthma guidelines for intensive care referral
    - deteriorating PEFR
    - persisting or worsening hypoxia
    - hypercapnia (or falling pH on arterial sample)
    - exhaustion, feeble respiration
    - reduced conscious level
    - respiratory arrest
- NIV
    - there may be a role for NIV in the management of life-threatening asthma, but this should only be done with a team ready to intubate and ventilate if there is deterioration
- intubation and ventilation
    - can be challenging in these patients and should be done by the most senior person (ideally consultant intensivist or anaesthetist)
- indications for admission
    - life-threatening features at any time
    - signs of severe asthma persisting after initial treatment

    - admission may be appropriate regardless of PEFR if patients have any of the following features:
        - still have significant symptoms
        - concern over compliance
        - lives alone
        - psychosocial problems
        - physical disability or learning difficulties
        - history of severe asthma
        - presentation at night
            - airways are most at risk around 0400
        - pregnancy
        - exacerbation despite adequate steroid dose pre-presentation
- patients suitable for discharge from the ED
    - patients should meet all of the following criteria:
        - no life-threatening features at any point (including pre-hospital)
        - no features of severe asthma after initial treatment
        - PEFR >75% of best or predicted and stable one hour after initial treatment OR PEFR >50% best or predicted and stable 2 hours after initial treatment
- discharge requirements
    - ideally a discharge checklist should be used
    - steroids
        - patients who initially had a PEFR of <50% best or predicted should be prescribed 40-50mg prednisolone PO for 5 days
        - if they have a steroid inhaler they should be advised to use it twice a day
        - a new inhaler should be prescribed if they have lost it, it is nearly empty or they have not previously used one
    - beta-2 agonist inhaler
        - check that the patient has an adequate supply with them
        - if not, or the inhaler is approaching empty, a new one should be prescribed
        - inhaler technique should be checked, and a spacer provided if technique is poor
    - GP/asthma nurse follow up
        - the patient should be told to see their GP in 48 hours or return to the ED earlier if symptoms worsen
        - a discharge letter should be sent to the GP by fax or email, and ideally an appointment made before the patient leaves the ED
    - lifestyle advice
        - smoking cessation advice
        - weight loss advice for obese patients – associated with improved symptom control
- pregnancy and breastfeeding
    - no drug treatment used in acute asthma has been demonstrated to be teratogenic
    - oxygen saturations should be maintained between 94% and 98%
    - obstetrician input is recommended in cases of severe asthma and continuous foetal monitoring commenced
    - drugs used in the treatment of acute asthma are excreted in breast milk, but in levels so minute they are not considered to pose a risk to the baby
        - breastfeeding should be continued

- NIV
    - advantages
        - helps overcome PEEPi (intrinsic positive end expiratory pressure) and decrease inspiratory work of breathing
        - can decrease expiratory work by opposing dynamic airway compression and allowing more expiration with less gas trapping and hyperinflation
        - reduces V/Q mismatch
        - significantly increases FEV1
        - significantly decreases hospital admissions
        - can be titrated to patient comfort
    - disadvantages
        - claustrophobia
        - agitation
        - gastric distention
        - dyssynchrony
        - increased expiratory work and hyperinflation
    - management
        - test patient comfort by starting with CPAP of 5cmH2O then titrate IPAP and EPAP to patient's comfort
- invasive ventilation
    - life-saving but associated with significant morbidity and mortality
    - indications
        - arrest
        - severe hypoxia
        - altered mental state
        - failure to respond to treatment
    - procedure
        - IV fluid loading and inotropes ready
        - large ET tube
        - RSI with ketamine/propofol
        - slow hand ventilation
        - attend to possible complications
    - ventilator settings
        - goal to avoid dynamic hyperinflation and hypoventilation
        - MV 100ml/kg/min (<8l/min in adult)
        - TV 6ml/kg
        - RR 10
        - I:E of 1:>4
        - PEEP at 60-80% of autoPEEP
        - adjust to degree of DHI (not CO2 or acidosis)
    - complications
        - hypotension and PEA arrest
            - causes
                - sedation
                - DHI
                - pneumothorax with tension
                - arrhythmias
                - hypovolaemia (rare)

        - endobronchial intubation
        - myocardial depression from prolonged hypoxia
        - reversal of pleural pressures impairing venous return
    - management
        - remove from ventilator
        - slow RR and load with fluid
        - auscultate the chest
        - check ETCO2 and ECG
        - urgent CXR
        - treat cause
        - fluids and inotropes
        - consider heliox and ECMO
- pneumothorax
    - decrease ventilation to protect other lung
    - decompress if hypotensive
    - if not, urgent CXR as signs not reliable

RC2 bronchiolitis

- background
    - inflammation of the small bronchi and bronchioles
    - caused by a viral infection
        - respiratory syncytial virus in up to 80%
        - other possible causative viruses include human metapneumovirus, adenovirus, rhinovirus, parainfluenza, influenza
    - in some cases there may be infection with more than one virus
- epidemiology
    - occurs in infants under 2 years
        - peaks between 3 months and 6 months
    - around a third of babies develop bronchiolitis before the age of 1 year
        - 2-3% require hospitalisation
    - peak incidence is in the winter months
- risk factors
    - environmental and social risk factors
        - older siblings
        - nursery attendance
        - passive smoke, particularly maternal
        - overcrowding
    - breastfeeding is considered protective
    - most admissions are in infants born at term with no risk factors
    - risk factors for severe disease/complications include:
        - prematurity (<37 weeks)
        - low birth weight
        - mechanical ventilation when a neonate
        - age less than 12 weeks
        - chronic lung disease
            - e.g. cystic fibrosis, bronchopulmonary dysplasia
        - congenital heart disease

        - neurological disease with hypotonia and pharyngeal discoordination
        - epilepsy
        - insulin-dependent diabetes
        - immunocompromise
        - congenital defects of the airways
        - Down's syndrome
- presentation
    - NICE guideline recommendations:
        - child under 2 years presenting with a 1-3 day history of coryzal illness followed by:
            - persistent cough AND
            - either tachypnoea or chest recession (or both) AND
            - either wheeze or crackles on chest auscultation (or both)
    - other typical features include fever (usually <39°) and poor feeding
    - very young babies may present with apnoea alone and no other signs
- differential
    - pneumonia or other pulmonary infections (e.g. mycoplasma, pertussis)
    - recurrent viral-triggered wheezing
    - meningitis
        - should always be considered as important not to miss
    - foreign body aspiration
        - rapid onset and failure to respond to initial management – high index of suspicion required
    - croup
        - usually stridor rather than wheeze
    - aspiration pneumonia
        - e.g. poor airway protection, seizures, cerebral palsy
    - GORD
    - asthma
    - chronic pulmonary disease
    - mediastinal mass
    - trachea-oesophageal fistula
        - late presentation with cough, abdominal distension and recurrent chest infections
    - congenital heart disease and heart failure
        - consider in neonates
    - vascular ring, congenital lobar emphysema, bronchogenic cyst
- assessment
    - history
    - examination
        - including central cap refill, respiratory rate, heart rate, chest signs
        - check oxygen saturations
    - consider admission for:
        - respiratory rate >60
        - inadequate fluid intake or signs of dehydration
        - under 3 months or born prematurely

        - comorbidity (particularly respiratory or heart disease or immunocompromise)
        - social circumstance and the ability of the carer to detect deterioration
    - admit if:
        - apnoea (observed or reported)
        - marked chest recession or grunting
        - respiratory rate >70
        - central cyanosis
        - oxygen saturations <92%
        - child looks unwell
- investigations
    - pulse oximetry
    - viral throat swabs
    - if deterioration (or high fever/focal chest signs), consider:
        - CXR
        - blood tests
        - blood gas
- management
    - mild cases
        - advise parents that illness is self-limiting, with symptoms worst on night 2-3
            - typically lasts 7-10 days
        - antipyretics if child is distressed or miserable with fever
        - cough can continue for 3-4 weeks
        - safety netting
    - patients requiring admission
        - admit:
            - patients <6 weeks
            - patients at risk of severe infection
            - sats <94%
                - dissociation curve is pushed to right in a febrile, acidotic child, so a higher PO2 is required to maintain the same level of saturation
        - supportive care
        - feeding/hydration
            - small, frequent feeds where tolerated
            - NG feeding where needed
            - IV therapy if severely unwell with nasal therapy or moderately dehydrated
                - aim for 2/3 maintenance to reduce risk of SIADH/hyponatraemia
                    - 0.9% saline and 5% dextrose preferred
        - oxygen when required
            - often via high flow nasal cannulae
                - 2l/kg/min provides PEEP of 4-8cm
            - CPAP if impending respiratory failure
        - no evidence for use of bronchodilators, steroids, nebulised adrenaline, hypertonic saline, antibiotics

                - reasonable to trial salbutamol if there is a strong family history or atopy or asthma and review afterwards
            - indications for intubation:
                - increasing work of breathing despite NIV
                - deterioration despite CPAP
                - not tolerating CPAP and continuing to desaturate
                - apnoea
                - transport
- prophylaxis
    - monoclonal antibody palivizumab recommended for:
        - patients with bronchopulmonary dysplasia due to prematurity or chronic lung disease
        - patients with congenital heart disease
        - patients with combined immunodeficiency syndrome

RC3 COPD

- background
    - estimated 3 million people with COPD in the UK
    - around 30,000 deaths/year
- definition
    - respiratory disease characterised by airflow obstruction that is not fully reversible
        - airflow defined as FEV1/FVC ratio of <0.7
    - diagnosis should be considered in any patient over 35 presenting with exertional breathlessness, cough, sputum production, wheeze or frequent winter bronchitis in the presence of risk factors
    - biggest risk factor is smoking
        - other risk factors include:
            - occupational exposure to fumes or dust
            - occupational exposure to tobacco smoke
            - alpha 1 antitrypsin deficiency
- pathophysiology
    - there is chronic airway inflammation
    - in an exacerbation there is an acute increase in inflammatory mediators which accelerate tissue damage
    - airway abnormalities include:
        - chronic inflammation with increased neutrophils, macrophages and T lymphocytes
        - increased number of goblet cells
        - mucus gland hyperplasia
        - fibrosis
        - narrowing and reduction of small airways
        - airway collapse
    - lung parenchymal abnormalities include abnormal dilation or destruction of the respiratory bronchiole or alveolar sac
    - in the pulmonary vasculature there is intimal hyperplasia and smooth muscle hypertrophy secondary to chronic hypoxic vasoconstriction
    - the changes cause increased airway resistance and expiratory airflow obstruction

        - increases the work of breathing and reduces minute ventilation
            - potentially leads to hypercarbia
        - airway destruction results in hypoventilation and ventilation/perfusion mismatch which causes hypoxaemia
- categories of severity
    - stage 1 – mild
        - post bronchodilator FEV1/FVC ratio <0.7
        - FEV1 >80% predicted
    - stage 2 – moderate
        - post bronchodilator FEV1/FVC ratio <0.7
        - FEV1 50-79% of predicted
    - stage 3 – severe
        - post bronchodilator FEV1/FVC ratio <0.7
        - FEV1 30-49% of predicted
    - stage 4 – very severe
        - post bronchodilator FEV1/FVC ratio <0.7
        - FEV1 <30% of predicted
- aetiology of exacerbations
    - 30% unknown aetiology
    - viruses
        - rhinovirus
        - influenza
        - parainfluenza
        - coronavirus
        - adenovirus
        - respiratory syncytial virus
        - chlamydia pneumoniae
    - bacteria
        - Haemophilus influenzae
        - Streptococcus pneumoniae
        - Moraxella catarrhalis
        - Staphylococcus aureus
        - Pseudomonas aeruginosa
    - pollutants
        - nitrogen dioxide
        - particulates
        - sulphur dioxide
        - ozone
    - other causes to consider
        - pneumothorax
        - pulmonary embolism
        - intra-abdominal pathology
        - cardiac failure
        - metabolic disturbance
- clinical assessment
    - history
        - breathlessness
        - cough

        - wheeze
        - chest tightness
        - increase or change in sputum production
        - inability to cope at home
        - reduced exercise tolerance
        - fatigue
        - confusion
        - ankle swelling
    - important aspects to consider:
        - past medical history including previous respiratory disease
        - medication history including recent steroid use
        - home oxygen use
        - nebuliser use
        - previous hospital admissions
        - use of any invasive or non-invasive ventilation
        - exercise tolerance – normal and current
        - social history, especially housing, stairs, mobility, care support
        - smoking history
            - ideally calculate number of pack years
            - pack years = number of packs smoked per day multiplied by the number of years the patient has smoked
                - 1 pack = 20 cigarettes
        - previous ITU admissions
    - examination
        - oxygen saturations, heart rate, respiratory rate, blood pressure, temperature
            - respiratory rate may slow in a tiring patient
        - peripheral signs of respiratory disease:
            - tar staining
            - palmar erythema
            - cyanosis
            - asterixis
        - increased work of breathing
            - use of accessory muscles
            - tripod posture
            - pursed lip breathing
        - reduced ability to speak in full sentences
        - presence or absence of cyanosis
        - chest wall deformity or barrel shaped chest secondary to hyperinflation
        - reduced chest wall movement
        - hyperresonance on percussion
        - auscultation – reduced air entry, wheeze, prolonged expiratory phase +/- crepitations
        - conscious level
        - signs of right heart failure such as raised JVP, peripheral oedema, hepatomegaly

        - BMI may be low due to reduced intake and calorie expenditure to increased work of breathing
        - sputum assessment
- investigations
    - at time of diagnosis
        - CXR to exclude other pathology
        - FBC – anaemia, polycythaemia
        - calculation of BMI
        - other possible investigations:
            - serial PEFR measurements if asthma is a differential
            - alpha 1 antitrypsin levels if early onset of symptoms, minimal smoking history or relevant family history
            - CT chest to investigate CXR abnormality or exclude PE
            - echo and ECG to assess for cor pulmonale
            - sputum culture in cases of chronic productive cough
    - in the ED when patient presents with exacerbation
        - ABG
            - acidosis
            - hypercapnia
            - hypoxaemia
            - chronic metabolic compensation
        - CXR
            - evidence of pneumothorax
            - consolidation
            - other pathologies that may cause increased breathlessness
        - ECG
            - exclude other or concurrent causes of breathlessness e.g. ischaemic heart disease, suggestion of PE
            - in severe disease there may be signs of pulmonary hypertension such as peaked p waves or right ventricular hypertrophy
        - FBC
            - rule out anaemia as cause of breathlessness
            - evidence of secondary polycythaemia
        - U&Es
        - theophylline level if patient on theophylline
        - sputum analysis – send for microscopy, culture and sensitivity if purulent
        - blood cultures if pyrexic
- risk stratification
    - option for treating at home:
        - able to cope at home
        - mild breathlessness
        - good general condition
        - good level of activity
        - no cyanosis
        - no worsening of peripheral oedema
        - normal level of consciousness
        - not already on long term O2

        - good social circumstances
        - no acute confusion
        - no rapid onset
        - no significant comorbidity
        - sats >90%
        - no CXR changes
        - arterial pH >7.35
        - arterial PaO2 >7 kPa
    - consider admission if any of these criteria are not met
- management
    - bronchodilators and oxygen
        - increased dose of short acting bronchodilators via inhaler or nebuliser as indicated
            - usually beta 2 agonists such as salbutamol or terbutaline and/or anticholinergics such as ipratropium bromide
            - increase dose and frequency of existing medication regime
            - nebulisers should be air driven if hypercapnic or acidotic
            - target saturations usually 88-92%
    - steroids
        - oral corticosteroids for all patients if breathlessness interferes with ADLs unless there are contraindications
            - reduces treatment failure, relapse and length of hospital stay and improves symptoms
        - prednisolone 30mg for 7-14 days
    - antibiotics
        - if increase in purulent sputum, consolidation on CXR or clinical signs of pneumonia
    - theophylline/aminophylline
        - consider IV only if inadequate response to nebulised bronchodilators
        - omit loading dose if patient on oral theophylline
        - reduce dose of oral theophylline if patient requiring macrolide or fluoroquinolone antibiotics
    - NIV
        - preferred initial mode of ventilation to treat acute respiratory failure
        - improves oxygenation and acidosis, decreases respiratory rate, work of breathing and severity of breathlessness
        - should be considered within 60 minutes of hospital admission when respiratory acidosis persists despite maximal medical management:
            - O2 to maintain sats 88-92%
            - nebulised salbutamol 2.5-5mg
            - nebulised ipratropium 500mcg
            - prednisolone 30mg
            - antibiotic agent (where indicated)
        - GOLD strategy 2018 indications for NIV include:
            - pH <7.35 and PaCO2 >6kPa
            - severe dyspnoea with clinical signs suggestive of muscle fatigue, increased work of breathing

- persistent hypoxaemia despite supplemental oxygen therapy
- inclusion criteria for NIV:
  - primary diagnosis of COPD exacerbation
  - able to protect airway
  - conscious and co-operative
    - can be considered for unconscious patients in critical care setting or if intubation inappropriate
  - patient's wishes considered and potential quality of life acceptable for patient
- exclusion criteria for NIV
  - life threatening hypoxaemia
  - severe co-morbidity
  - confusion/agitation/cognitive impairment
  - facial burns/trauma/ recent facial or upper airway surgery
  - vomiting
  - fixed upper airway obstruction
  - undrained pneumothorax
  - upper gastrointestinal surgery
  - inability to protect the airway
  - copious respiratory secretions
  - haemodynamically unstable requiring inotropes/vasopressors (unless in critical care unit)
  - patient moribund
  - bowel obstruction
  - patient declines treatment
- starting NIV
  - clear plan of escalation and ceiling of care documented in notes
  - patient in semi-recumbent position, with full face mask for first 24 hours, switched to nasal if preferred by patient
  - initial IPAP of 10 cmH2O and EPAP of 4-5 cmH2O should be used
    - should be increased at around 5 cmH2O every 10 minutes to an IPAP target of 20 cmH2O, or patient unable to tolerate further, or therapeutic response achieved
  - if the patient benefits within the first 4 hours it should be continued for as long as possible
- monitoring NIV
  - ABG at baseline, 1 hour and 4 hours after commencing and 1 hour after changing any settings
  - continuous pulse oximetry and ECG monitoring for first 12 hours

- indications for invasive mechanical intervention
  - decision should be made within 4 hours of starting NIV as improvements should be apparent by then
  - indications include:
    - inability to tolerate NIV or NIV failure
    - post respiratory or cardiac arrest
    - diminished consciousness, psychomotor agitation inadequately controlled by sedation

                - massive aspiration or persistent vomiting
                - persistent inability to remove respiratory secretions
                - severe ventricular or supraventricular arrhythmias
                - severe haemodynamic instability unresponsive to fluid and vasopressors
                - life-threatening hypoxaemia in patients unable to tolerate NIV
        - other management
            - hospital at home and assisted discharge schemes should be considered if feasible
            - patients should be advised about and assisted with smoking cessation
- prognosis
    - predictors of mortality include:
        - increasing age
        - significant comorbidity
        - decreasing post-bronchodilator FEV1
        - patients already on long term oxygen therapy
        - increased number of COPD exacerbations, particularly 3 or more episodes
        - increased number of hospital admissions
        - poor performance status
        - low arterial pH on admission
        - presence of bilateral leg oedema
        - home circumstances, particularly if in nursing home
        - unrecordable peak flow on admission
        - pulse oximetry showing sats <86%
        - intervention with assisted ventilation

RC4 foreign body inhalation

- background
    - most common in young children (1-3 years)
    - consider in any child with respiratory symptoms
    - 20% lodged in upper airway, 80% in bronchus
- clinical features
    - history of eating or handling a small object (or being unsupervised near one) followed by sudden onset of cough or dyspnoea
        - onset of respiratory symptoms sometimes delayed by >24 hours
    - dyspnoea, tachycardia, respiratory distress, hypoxia
    - sudden collapse, cardiac arrest
    - cough, gagging
    - stridor, dysphonia (if stuck at level of larynx)
    - wheeze, decreased breath sounds (if lower airways)
        - wheezing is not responsive to bronchodilators
    - fever and pneumonia symptoms if retained
- evaluation
    - CXR
        - useful to confirm diagnosis but does not rule out
            - negative in >50% of tracheal foreign bodies and 25% of bronchial foreign bodies

                - may be missed if very small and/or radio-opaque
            - inspiratory/expiratory films may show relative hyperinflation proximal to obstruction on end expiration
            - consider lateral decubitus views
- management
    - complete airway obstruction
        - back blows, Heimlich, chest thrusts
        - remove object if visible (caution to not push back into airway)
        - laryngoscopy and removal of object with Magill's forceps
        - if unsuccessful, bag valve mask ventilation and/or intubation
            - may dislodge object and improve situation to partial or more distal obstruction
        - if unable to intubate, may need cricothyrotomy (will not help if obstruction distal to cricothyroid)
    - partial airway obstruction
        - supplemental oxygen
        - allow patient to assume most comfortable position
        - monitor closely
        - may need rigid bronchoscopy to remove
        - may need ENT and anaesthetics
            - inhalational induction is preferred over IV – less risk of foreign body moving distally, which makes it easier to remove
                - however depth of anaesthesia and increased resistance to ventilation with scope in increases risk of hypercarbia
            - IV induction immobilises the airway and decreases atelectasis
                - however access can be difficult and may precipitate decompensation
        - post-removal, consider dexamethasone, bronchodilators and/or adrenaline and antibiotics for pneumonia
- complications
    - infection
    - bleeding
    - pneumothorax
    - airway trauma and swelling
    - tracheal perforation

RC5 pertussis

- background
    - cause by the bacterium Bordetella pertussis
        - small Gram-negative coccobacillus
    - greatest risk of complications in infants under 6 months
    - can occur in immunised children but generally less severe
    - patients are infectious just prior to onset and until 21 days after onset of cough if untreated
- history
    - classic whooping cough
        - catarrhal phase

                - cough and coryza for one week
            - paroxysmal phase
                - more pronounced cough in spells/paroxysms
    - can present as non-specific persistent cough
    - vomiting often follows coughing spasm
    - infants may develop apnoea and/or cyanosis with coughing spasms
    - close contact with a case of pertussis may raise suspicion
    - other family members often have a cough (>70% of household contacts are also infected)
- examination
    - often no clinical signs
    - children are usually well between coughing spasms
    - observation of coughing spasms may be helpful
    - fever is uncommon
    - subconjunctival haemorrhages may be seen
- investigations
    - diagnosis is clinical so laboratory confirmation not required, however it can be helpful for infection control
    - oral fluid testing for anti-pertussis toxin IgG
    - nasopharyngeal aspirate/swab for culture or PCR
        - PCR test is usually negative after 21 days or 5-7 days after effective antibiotic therapy started
    - pertussis serology (IgA) may be detectable 2 weeks after the onset of illness but rarely affects clinical management
- management
    - consider antibiotics if:
        - diagnosed in catarrhal or early paroxysmal stage
            - may reduce severity
        - cough for less than 14 days
            - may reduce spread, reduces school exclusion period
        - admitted to hospital
        - complications (pneumonia, cyanosis, apnoea)
        - choice of antibiotic:
            - clarithromycin for babies <1 month
            - azithromycin or clarithromycin for children 1 month or older and non-pregnant adults
            - erythromycin for pregnant women
            - co-trimoxazole is an alternative to macrolides
    - exclude from school and contact with others outside the home until received 5 days of therapy or coughing for more than 21 days
    - unimmunised or partially vaccinated patients diagnosed with pertussis should still complete the vaccination schedule
    - all suspected or confirmed cases must be notified
- prophylaxis
    - antibiotic prophylaxis required if:
        - close contact with confirmed case whilst they were infectious AND
        - first contact was within 14 days (or 21 days for infants <6 months) AND

        - children
            - age <6 months OR
            - <3 doses pertussis vaccine OR
            - household member <6 months OR
            - attend childcare in same room as infant <6 months
        - adults (regardless of immunisation status)
            - expectant parents in last month of pregnancy OR
            - healthcare worker in maternity hospital or newborn nursery OR
            - childcare worker in close contact with infants <6 months OR
            - household member <6 months
    - booster vaccination should be considered for adults not receiving a dose in the past 10 years
- complications
    - pneumonia
    - apnoea
    - seizures
    - encephalitis
    - otitis media
    - dehydration
    - weight loss
    - problems caused by raised intra-thoracic or intra-abdominal pressure during coughing such as:
        - fractured ribs
        - subconjunctival haemorrhage
        - inguinal or other hernia
        - rectal prolapse
        - pneumothorax
        - urinary incontinence
        - petechiae

RC6 pleural effusion

- background
    - abnormal collection of fluid within the pleural space
    - annual incidence in the developed world is around 320 per 100,000 population
    - often found incidentally in the ED
    - morbidity and mortality is related more to the underlying cause than the size of the effusion
- normal anatomy and physiology
    - the pleural space is walled by the parietal pleura lining the inside of the chest wall and visceral pleura lining the lung
    - pleural fluid is produced by filtering from systemic capillaries within the parietal interstitium
        - production is greatest at the apex of the lung
    - fluid enters the pleural space down a small pressure gradient
    - there is around 0.13ml/kg fluid in healthy people to allow smooth movement of the lungs during normal ventilation

- pleural fluid is absorbed and drained via parietal lymphatic vessels mainly in the region of the diaphragm and mediastinum
    - they ultimately drain into the mediastinal lymph nodes
    - the visceral pleura plays an insignificant part in pleural fluid production and turnover
- normal fluid physiology
    - volume is maintained by the balance of pulmonary capillary hydrostatic and oncotic pressure, lymphatic drainage and the integrity of the pleural and capillary membranes
        - disturbances to any of these can lead to formation of excess pleural fluid
    - normal pleural fluid is formed from the filtration of plasma by healthy parietal pleural membranes, and has the following characteristics:
        - glucose content similar to plasma
        - low sodium content (1-2g/dl)
        - low white cell count (<1000 cells/mm^3)
        - low lactate dehydrogenase level (<50% that of plasma)

- pathophysiology
    - transudates
        - associated with increased systemic or pulmonary capillary hydrostatic pressure or decreased colloid osmotic pressure (often co-existent)
        - the pleural membranes are intact and the permeability of pleural capillaries to proteins is normal
        - causes include:
            - cardiac failure
            - liver failure/cirrhosis
            - hypoalbuminaemia
            - peritoneal dialysis
            - renal failure/nephrotic syndrome
            - mitral stenosis
            - pulmonary embolism
            - constrictive pericarditis
            - hypothyroidism
            - urinothorax
            - SVC obstruction
            - ovarian hyperstimulation
            - Meig's syndrome (right pleural effusion with ovarian fibroma)
    - exudates
        - associated with altered permeability of pleural membranes, increased capillary wall permeability to proteins or vascular disruption
        - can also be associated with reduced or obstructed lymphatic drainage from the pleural space
        - causes include:
            - pneumonia
            - malignancy
            - viral disease
            - CABG surgery

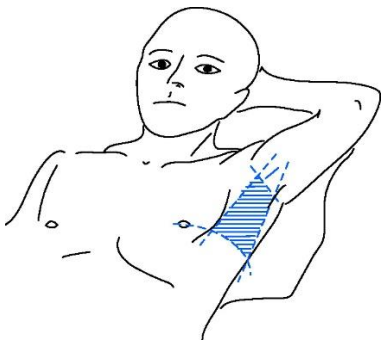
                - pulmonary infarction
                - rheumatoid arthritis
                - autoimmune disease
                - benign asbestos effusion
                - pancreatitis
                - post MI (Dressler's syndrome)
                - yellow nail syndrome
                - drugs
                - fungal infection
        - effusions can also be caused by aortic dissection, oesophageal rupture and pancreatitis
        - effusion in the context of trauma should be assumed to be haemothorax until proved otherwise
        - malignant effusions
            - tumours most commonly associated with malignant pleural effusions are:
                - lung
                - breast
                - lymphoma
                - tumours of the GI tract
            - tumour cells migrate to the pleura via haematogenous or lymphatic spread
            - tumour cells disrupt the network of lymphatic channels within the parietal pleura, causing obstruction
                - effusions associated with malignancy are therefore exudates
        - empyema
            - simple parapneumonic effusions can become complex parapneumonic effusions then empyemas
            - more than half of patients with pneumonia develop parapneumonic effusions – empyema results when the fluid becomes infected
            - primary empyemas can occur in patients without underlying pneumonia
            - empyemas require drainage
            - aerobic organisms are most common
                - common Gram positives include streptococcal and staphylococcal species
                - common Gram negatives include E. coli, Pseudomonas spp, Haemophilus influenzae and Klebsiella spp
            - the most common associated underlying diseases are:
                - diabetes mellitus
                - malignancy
                - COPD
                - CNS disease
            - approximately a third of patients with empyema have no underlying health problems
- history
    - symptoms due to effusion
        - dyspnoea
        - chest pain that may be pleuritic
        - cough

        - shoulder pain
        - lethargy
    - symptoms due to the underlying cause
        - weight loss
        - haemoptysis
        - fever
        - night sweats
        - swollen legs/abdomen
    - specific history points:
        - heart, liver or renal disease
        - history of malignancy (however remote)
        - chest disease, especially recent pneumonia
        - pancreatic disease
        - connective tissue disease
        - previous cardiac surgery
        - smoking history
        - occupational history, especially asbestos exposure
        - previous TB exposure and HIV status
        - risk factors for VTE
        - recent trauma, especially if anticoagulated (suspicion of haemothorax)
        - accurate drug history
            - drugs known to cause pleural effusions include:
                - phenytoin
                - methotrexate
                - amiodarone
                - nitrofurantoin
                - beta-blockers
            - recent reduction in diuretics
- examination
    - signs due to pleural effusion
        - respiratory distress
        - dyspnoea
        - hypoxia
        - asymmetrical lung expansion
        - reduced breath sounds
        - reduced tactile vocal fremitus
        - stony dull to percussion
        - tracheal deviation
    - signs due to underlying cause
        - clubbing
        - cachexia
        - lymphadenopathy
        - peripheral oedema
        - raised JVP
        - ascites
        - peripheral stigmata of cardiac/renal/liver disease
        - previous operative scars or radiotherapy marks
        - signs of DVT

        - tar staining
        - signs of connective tissue disease
- imaging
    - CXR
        - blunting of the costophrenic angle is visible with 250-500ml of fluid accumulation
        - there should be a meniscus – if it is completely flat it may suggest a concurrent pneumothorax
    - US
        - can provide rapid bedside diagnosis
        - recommended for all therapeutic aspirations and chest drain insertion
    - CT
        - aids with identification and quantification of effusions
        - can help identify causative underlying pathology
        - may be used by interventional radiologists for difficult drainage procedures and planning biopsies
    - bloods
        - ABG
            - if concerns about oxygenation/ventilation
        - FBC and CRP
            - assess inflammatory response
        - U&E, LFTs
            - identify renal or live disease
        - serum protein, LDH and glucose
            - for Light's criteria
        - serum amylase
            - if any suspicion of pancreatitis or ruptured oesophagus
        - d-dimer
            - if PE suspected with low Well's score
- pleural fluid
    - appearance
        - putrid odour
            - anaerobic empyema
        - food particles
            - ruptured oesophagus
        - bile stained
            - biliary fistula (cholothorax)
        - milky
            - chylothorax
        - 'anchovy sauce'
            - ruptured amoebic abscess
    - analytic tests
        - protein
            - transudate contains <25g/L protein
            - exudate contains >35g/L protein
        - LDH
        - cytology, cell count and differential

        - fluid pH
        - fluid glucose
        - Gram staining and culture +/- acid fast bacilli
        - haematocrit (if uncertainty about presence of haemothorax
    - Light's criteria
        - used if protein in pleural fluid between 25 g/L and 35 g/L
        - fluid is exudate if one or more of:
            - pleural fluid: serum protein ratio >0.5
            - pleural fluid LDH: serum LDH >0.6
            - pleural fluid LDH >two thirds the upper limit of normal serum LDH
    - cytology
        - malignant effusions can be diagnosed by cytology in around 60% of cases
    - cell count/differential
        - high neutrophils
            - pulmonary embolism
            - pancreatitis
            - pneumonia
            - empyema
        - high lymphocytes
            - malignancy
            - TB
            - pleuritis
        - eosinophilia
            - pneumothorax
            - haemothorax
            - asbestosis
            - Churg-Strauss
        - mononuclear cells
            - chronic inflammatory process
    - pleural fluid pH
        - should be measured on all non-purulent effusions
            - pus is an indication for drainage regardless of pH and may damage the machine
        - can be done using heparinised sample of fluid in standard blood gas analyser
        - low pH
            - infection
            - malignancy
            - oesophageal rupture
            - rheumatoid disease
        - fluid pH of <7.2 in infection indicates tube drainage required and differentiates between simple and complex para-pneumonic effusion
    - pleural fluid glucose
        - low glucose is found in the same conditions as low pH
        - lowest levels found in rheumatoid arthritis and empyema
    - amylase
        - useful in suspected oesophageal rupture or pancreatitis – levels will be higher than normal serum amylase

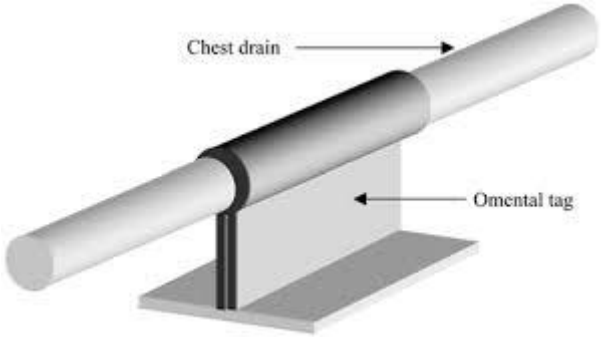
    - triglyceride and cholesterol levels
        - may be useful if chylothorax suspected
- management
    - no national consensus on which patients should undergo diagnostic aspiration, therapeutic aspiration or drainage in the ED
    - take into account local guidelines and the condition of the patient
    - BTS guidelines state that aspiration should not be performed for bilateral effusions in a clinical setting strongly suggestive of a transudate unless there are atypical features or failure to respond to therapy
        - focus for suspected transudate should be on identifying and treating the underlying diagnosis
    - most common indication for drainage in the ED is large effusion causing significant hypoxia or distress, particularly with mediastinal shift
    - if aspiration or drainage required:
        - unless the patient has significant cardiovascular or respiratory compromise, the procedure should not be performed overnight
        - full aseptic technique
        - non-urgent interventions should be avoided in anticoagulated patients until INR <1.5 (caution in DOACs)
        - recent CXR should be reviewed prior to insertion
        - bedside US should be used to increase likelihood of success and reduce risk of complications
        - suitably trained practitioner should perform or directly supervise the procedure
        - consent should be gained unless it is an emergency – ideally in writing if a drain is being inserted
        - there must be suitable and clearly organised follow up for investigations requested in the ED
    - aspiration and drainage:



https://www.rcemlearning.co.uk/reference/pleural-effusion/#1571046547311-bfdedfe9-cc62

        - preferred site is within the triangle of safety, bordered by
            - anteriorly, lateral edge of pectoralis major
            - laterally, lateral edge of latissimus dorsi
            - inferiorly, line of fifth intercostal space
            - superiorly, base of axilla
        - posteriorly lying effusion may be best approached with imaging guidance from the back and patient sitting forward

- diagnostic aspiration
    - 21G (green) needle attached to 50ml syringe directed along the top of a rib
    - avoid medial sites (<5cm from thoracic spine) as the intercostal artery is in the middle of the intercostal space in this region
    - local anaesthetic not required if landmarks easily felt
    - CXR not required after simple pleural aspiration unless air is withdrawn, the procedure is difficult, multiple attempts are required or the patient becomes symptomatic
- therapeutic aspiration
    - 1% lignocaine infiltrated to skin and particularly the pleura
    - cannula attached to three way tap and tubing/syringe
    - 20G or smaller cannula where feasible
        - weak evidence that large bore cannula more likely to cause pneumothorax
        - larger needle may be required for thick chest walls
    - procedure should be stopped when no more fluid or air can be aspirated, the patient develops symptoms of a cough or chest discomfort or 1.5 litres have been withdrawn
    - a simple dressing should be applied after the cannula is withdrawn
    - when cause is unclear and effusion large, a post-procedure CXR may be done to view the underlying lung
- chest drainage
    - antibiotic prophylaxis not recommended for non-trauma patients
    - local anaesthetic and analgesia should be used
    - procedural sedation should be considered
    - needle aspiration following local anaesthetic infiltration should confirm the presence of fluid before proceeding to drain insertion
    - never use substantial force for insertion of drain
    - dilator should not be inserted >1cm beyond the depth from skin to pleural space
    - small drains (8-14Fr) should be used for free flowing effusions or infections
    - drain should be secured using sutures and omental tag of tape



https://journals.sagepub.com/doi/abs/10.1191/1460408605ta341oa?journalCode=traa

    - should be connected to underwater drainage system and no more than 1.5 litres allowed to drain in first hour after insertion
    - repeat CXR after insertion

        - drain may be withdrawn to correct malposition but should not be inserted further due to risk of infection
- parapneumonic effusions
    - simple effusions usually respond to antibiotics and do not require drainage
    - effusions are complex, and require drainage, if:
        - low fluid pH and glucose or
        - elevated WCC +/- organisms
    - intra-pleural fibrinolytic drugs may be given if the effusion does not respond to drainage, or surgical referral if this fails
- malignant effusions
    - usually treated with pleurodesis (e.g. intrapleural tetracyclines, talc, bleomycin) to reduce likelihood of recurrence
    - some patients may need intrapleural fibrinolytic therapy (e.g. urokinase) for fibrous or loculated effusions
    - other management includes thoracoscopy, intermittent therapeutic drainage (usually for short life expectancy), long term indwelling pleural catheter, pleuro-peritoneal shunting

RC7 pneumonia

- community acquired pneumonia in adults
    - BTS and NICE guidelines both available
        - not applicable to:
            - children
            - patients with COPD
            - immunosuppressed patients
            - inpatients
            - patients discharged in the preceding 10 days
    - background
        - CAP occurs in 0.5-1% of UK adults/year
        - mortality low for community-treated patients (<1%) but >5% for patients requiring admission and >30% for patients requiring ICU
        - one of the commonest causes of in-hospital death
        - more than half of deaths are in patients >84 years
    - history
        - fever
        - productive cough
        - sweating
        - shivering
        - myalgia
        - dyspnoea
        - pleuritic chest pain
        - factors that would influence management plan:
            - prior or current antibiotic use
            - allergies
            - current medications
            - known respiratory disease (e.g. COPD)
            - co-morbidities

                - immunosuppression
                - social circumstances
                - smoking status
                - age >50
                - new confusion reported by friend, relative or carer
            - questions about foreign travel and contact with animals are unlikely to be helpful, except in severe illness (where frequency of legionella and staphylococcal infection is higher)
    - organism specific features (not specific enough to influence initial antibiotic choice)
        - Streptococcus pneumoniae
            - increasing age
            - co-morbidity
            - acute onset
            - high fever
            - pleuritic chest pain
        - Legionella pneumophila
            - younger patients
            - smokers
            - absence of co-morbidity
            - diarrhoea
            - neurological symptoms
            - more severe infection
            - evidence of multisystem involvement
            - cases peak in the autumn in the UK and 50% are related to travel abroad; contaminated water cooling systems can cause clusters
        - Mycoplasma pneumoniae
            - younger patients
            - less multisystem involvement
            - epidemics occur in the UK spanning 3 winters every 4 years
        - Staphylococcus aureus
            - incidence peaks in winter
            - more common in intravenous drug users/debilitated patients, particularly those with influenza
            - relatively more prevalent in patients on intensive care
                - still less common than Strep pneumoniae
    - examination
        - cardio-respiratory examination
            - bronchial breathing
            - dullness to percussion
            - crepitations
            - pyrexia
            - tachypnoea
            - tachycardia
            - findings pointing to alternative diagnosis such as pulmonary oedema, pleural effusion or pneumothorax
    - investigations

- CXR
    - most useful test in the ED
    - can confirm diagnosis, exclude alternatives such as pneumothorax or identify alternative diagnoses such as pulmonary oedema
    - recommended by BTS for all patients being admitted from the ED to confirm or refute diagnosis
    - not required on patients being discharged with a diagnosis of CAP unless diagnosis is in doubt or patient is considered at risk of other pathology such as lung cancer
    - CXR changes include:
        - areas of consolidation, usually limited to one lobe but may involve multiple lobes or both lungs
        - small pleural effusions are common and usually reactive
        - AP resus films can be of poor quality and make it difficult to distinguish between pulmonary oedema and pneumonia
        - early CXRs may be normal
        - signs that may help to confirm diagnosis of pneumonia include:
            - silhouette sign
                - loss of the normal border between structures
                    - right heart margin lost in middle lobe pneumonia
                    - border of diaphragm obscured in right lower lobe pneumonia whilst right heart margin remains clear
                    - left lower lobe pneumonia may be missed if the area behind the cardiac shadow is not routinely examined
            - air bronchogram
                - as the bronchi branch a point is reached where the walls are difficult to visualise unless calcified and it is not possible to distinguish air in the bronchi from air in the surrounding alveoli
                - if the surrounding alveoli fill with fluid or pus, branching radiolucent air passages (air bronchograms) may be seen
- CT
    - not helpful for diagnosis of pneumonia in the ED
- blood tests (BTS recommendations) for patients admitted with CAP
    - FBC
        - WCC >15 x10^9/L suggests bacterial pneumonia
    - U&Es
        - urea used for CURB-65 score

        - may also identify unsuspected renal failure or hyponatraemia
    - CRP
        - not useful diagnostically or prognostically on admission
        - forms baseline for later comparison
            - failure to fall after 72 hours suggests treatment failure
    - ABG
        - if oxygen saturations <94%
    - lactate
        - to identify tissue hypoperfusion in patients who are not yet hypotensive but at risk of septic shock
        - raised lactate (particularly >4) is poor prognostic factor
- microbiology
    - blood cultures
        - low sensitivity in CAP
            - particularly with non-severe CAP, no co-morbid disease or antibiotics prior to admission
        - BTS recommends blood cultures for moderate or severe CAP and most patients admitted with CAP, preferably before starting antibiotic therapy
        - blood cultures may be omitted if:
            - diagnosis of CAP definitely confirmed AND
            - patient has low severity pneumonia AND
            - patient has no significant co-morbid disease
    - sputum culture
        - purulent sputum should be sent for culture and sensitivity if:
            - severe pneumonia
            - moderate pneumonia without prior antibiotics
            - failure to improve with treatment
            - patients who test positive for urine legionella antigen
        - samples sent later from the ward will have lower yield due to ED antibiotic administration
- serology
    - may be done later, not usually required in ED
    - pneumococcal urine antigen
        - should be done for all patients with moderate or severe CAP
    - legionella urine antigen
        - should be sent for patients with severe pneumonia, specific risk factors and all patients during epidemics
        - NICE recommends testing patients with moderate or severe pneumonia
- PCR
    - may be selectively requested by respiratory physicians
    - available for mycoplasma pneumonia, Chlamydophila and respiratory viruses

        - ECG
            - pneumonia not associated with any specific ECG changes
            - sinus tachycardia common
            - pneumonia may trigger atrial fibrillation or rate related ischaemia in the elderly
    - risk stratification
        - CURB-65 score (1 point for each):
            - **c**onfusion
                - new onset disorientation in time, place or person OR AMT <8/10
            - **u**rea >7mmol/L
            - **r**espiratory rate >30
            - **b**lood pressure: systolic <90mmHg or diastolic <60mmHg
            - age >**65** years
        - AMTS
            - age
            - time (nearest hour)
            - address (repeat now and at end of test)
            - year
            - name of this place
            - identify 2 persons (e.g. doctor, nurse)
            - DOB
            - date of first world war
            - name of present monarch
            - count backwards 20-1
        - CURB-65 interpretation
            - mortality rises exponentially with increased score
                - 0 – 0.7% 30 day mortality
                - 1 – 3.2%
                - 2 – 13%
                - 3 – 17%
                - 4 – 41.5%
                - 5 – 57%
            - BTS classifies 3 or more as severe pneumonia and recommends admission
                - hospital treatment should be considered for patients with a score of 2 but outpatient management is acceptable in certain cases
            - most patients with a score of 0 or 1 can be managed in the community
        - other factors suggesting need for admission:
            - hypoxaemia regardless of FiO2 (SaO2 <94% or PaO2 <8 kPa)
            - bilateral or multi-lobe involvement on CXR
            - presence of co-existing disease such as congestive cardiac failure, chronic renal failure
            - age over 50
            - admission required for social reasons

- management
  - general measures
    - rest
    - plenty of oral fluids
    - stop smoking
    - if discharged, advise to see GP for review within 48 hours, or sooner if clinically indicated
  - specific management
    - antibiotics
      - follow local guidelines
      - start in the ED as soon as possible after diagnosis made – should be started within 4 hours of arrival
      - low or moderate severity should have oral antibiotics if able to swallow
        - amoxicillin 500mg TDS for 1 week
        - clarithromycin 500mg BD for 1 week if penicillin allergic
        - levofloxacin 500mg OD if allergic to both of these
      - patients with severe pneumonia or unable to swallow should be given IV antibiotics
        - co-amoxiclav 1.2g IV TDS plus clarithromycin 500mg BD IV for 7-10 days as first line treatment unless local guidelines state otherwise
        - consider cefuroxime plus clarithromycin if penicillin allergic
        - consider levofloxacin if allergic to both
    - oxygen
      - should be prescribed and administered if sats <94% on air or PaO2 <8kPa
    - steroids should not be used for pneumonia
    - chest physiotherapy
      - not routinely recommended, may have a role for those with underlying chest conditions
    - NIV
      - CPAP/NIV not recommended for respiratory failure secondary to pneumonia, no evidence of benefit
    - parapneumonic effusions
      - thoracocentesis recommended to identify those that are infected (empyemas)
      - empyemas require pleural drains
  - follow up
    - letter should be sent to GP recommending repeat CXR 6 weeks after completion of treatment if:
      - persistence of symptoms and signs
      - higher risk of malignancy (especially smokers and age >50)
- hospital-acquired pneumonia
  - background

        - new infection of lung parenchyma appearing more than 48 hours after admission to hospital
            - mostly occurs in patients who are severely debilitated, immunocompromised or mechanically ventilated
        - usually caused by S. pneumoniae if less than 5 days after admission
            - after this, it is usually caused by H. influenzae, MRSA, Pseudomonas aeruginosa and other Gram-negative bacteria
        - often caused by multiple organisms
    - investigations are as for CAP
    - antibiotics should follow local guidance
- aspiration pneumonia
    - background
        - results from inhalation of stomach contents or oropharynx secretions leading to lower respiratory tract infection
        - aspiration may cause:
            - chemical pneumonitis
                - chemical irritation of the lungs
                - may progress to acute respiratory depress syndrome and/or bacterial infection
            - obstruction of the respiratory tract from large volumes of aspirated material
            - bacterial infection
                - may lead to empyema, lung abscess, acute respiratory failure and acute lung injury
        - usual site is the apical and posterior segments of the lower lobe of the right lung
            - may include the posterior segment of the upper lobe if patient is supine
    - pathogens
        - often the normal flora of the oropharynx, including:
            - Strep. pneumoniae
            - Staph. aureus
            - Haemophilus influenzae
            - anaerobes
            - Streptococcus milleri group
            - Klebsiella pneumoniae (particularly with history of alcohol abuse)
        - pathogens of nosocomial aspiration pneumonia include:
            - oral anaerobes as above
            - Gram-positive cocci e.g. Peptostreptococcus spp
            - Gram-negative bacilli e.g. enterobacteria, Pseudomonas aeruginosa
            - MRSA
    - risk factors
        - impaired consciousness
            - drug or alcohol misuse
            - general anaesthesia
            - seizures
            - sedation

            - acute stroke
            - CNS lesions
            - head injury
        - swallowing disorders
            - oesophageal stricture
            - dysphagia
            - stroke
            - bulbar palsy
            - pharyngeal disease (e.g. malignancy)
            - neuromuscular disorders (e.g. MS)
        - poor mobility
        - nil by mouth status
        - increasing age
        - COPD
        - male gender
        - increasing number of medications
        - other:
            - trachea-oesophageal fistula
            - ventilator-associated pneumonia
            - periodontal disease
            - gastro-oesophageal reflux
            - post-gastrectomy
            - tracheostomy
- presentation
    - non-specific symptoms
        - fever
        - headache
        - nausea
        - vomiting
        - anorexia
        - myalgia
        - weight loss
    - cough
    - dyspnoea
    - pleuritic chest pain
    - purulent sputum
    - signs:
        - tachycardia
        - tachypnoea
        - decreased breath sounds
        - dullness to percussion
        - pleural friction rub
        - hypoxia and septic shock in severe infection
- management
    - mechanical obstruction
        - removal of object, usually by bronchoscopy
    - tracheal suction if seen early

        - may need intubation with positive pressure ventilation
        - bacterial infection of lower airways:
            - empirical antibiotics while awaiting culture results
            - community acquired aspiration pneumonia often treated initially with oral amoxicillin if low severity
                - for high severity, IV co-amoxiclav with clarithromycin or erythromycin
            - if hospital acquired, generally co-amoxiclav for non-severe infections and Tazocin for severe infections
        - uncertain role of steroids
        - supportive treatment with fluid management, bronchodilators, physiotherapy
        - referral to SALT
    - prognosis
        - depends on underlying cause, general well-being of patient, presence of complications, speed of diagnosis, effective treatment
- LRTI in children
    - background
        - infection below the level of the larynx, includes, bronchiolitis, bronchitis and pneumonia
        - estimated incidence is 30 per 1000 per year in the UK
    - pathophysiology
        - no universally agreed definition of LRTI
        - essentially is inflammation of the airways/pulmonary tissue due to viral or bacterial infection below the level of the larynx
            - GORD may cause chemical pneumonitis
            - smoke and chemical inhalation may cause pulmonary inflammation
        - viral infections
            - 45% of hospitalised children
                - Influenza A
                - RSV
                - human metapneumovirus
                - varicella zoster virus
        - bacterial infections
            - around 60% of hospitalised cases
                - Strep. pneumoniae (majority of cases)
                - H. influenzae
                - Staphylococcus aureus
                - Klebsiella pneumoniae
                - enterobacteria (e.g. E. coli)
                - anaerobes
        - atypical organisms
            - Mycoplasma pneumoniae (14% of hospitalised cases)
            - Legionella pneumophila
            - Chlamydophila pneumoniae (9% of hospitalised cases)
            - Coxiella burnetiid
        - secondary bacterial infection following viral URTI or LRTI relatively common

- presentation
  - consider bacterial pneumonia in patients with persistent or repetitive fever above 38.5° with chest recession and raised respiratory rate
    - audible wheezing is not often seen in LRTI
    - stridor or croup suggests URTI, epiglottitis or foreign body inhalation
  - history
    - newborns/neonates
      - grunting
      - poor feeding
      - irritability or lethargy
      - sometimes tachypnoea
      - fever (but neonates may be hypothermic)
      - cyanosis (in severe infection)
      - cough (unusual at this age)
      - beware of streptococcal sepsis and pneumonia in first 24 hours of life and chlamydial pneumonia (possibly accompanied by conjunctivitis) in the second or third week
    - infants
      - cough (most common symptom after first four weeks)
      - tachypnoea (according to severity)
      - grunting
      - chest indrawing
      - feeding difficulties
      - irritability and poor sleep
      - breathing may be described as wheezy but is usually upper airway noise
      - history of preceding URTI (very common)
      - atypical and viral infections in this age group may have low grade or no fever
    - toddlers/preschool children
      - preceding URTI is common
      - cough is the most common symptom
      - fever occurs mainly with bacterial organisms
      - pain (chest and abdomen) more common in this age group
      - vomiting after coughing is common
      - be aware that lower lobe pneumonias can cause abdominal pain and severe infections will compromise breathing more
    - older children
      - additional symptoms will be reported
      - they will be able to describe constitutional symptoms better
      - atypical organisms are more likely in older children
- examination
  - pulse oximetry
  - high fever (>38.5° common)
  - look for other diseases such as rashes, pharyngitis
  - signs of respiratory distress
    - cyanosis in severe cases

                - grunting
                - nasal flaring
                    - can be useful indicator of pneumonia in children under 12 months
                - marked tachypnoea
                - chest indrawing (intercostal and suprasternal recession)
                - subcostal recession
                - abdominal 'see-saw' breathing
                - tripod positioning
            - reduced sats <95%
    - markers of severe LRTI requiring admission
        - oxygen saturations <92%
        - RR >70 (or 50 in older child)
        - significant tachycardia for level of fever
        - cap refill >2sec centrally
        - difficulty in breathing, e.g. intermittent apnoea, grunting, not feeding
    - other reasons for admission include:
        - presence of co-morbidities
        - children under 6 months
        - failure to improve with 48 hours of antibiotics as outpatient
        - troublesome pleuritic pain

RC8 pneumothorax

- types of pneumothorax
    - primary
        - spontaneous pneumothorax in a healthy person
    - secondary
        - associated with underlying lung disease
            - greater consequences and more difficult to manage
            - BTS automatically considers long term smokers over 50 to have secondary pneumothoraces
            - causes include
                - obstructive airways disease
                    - asthma
                    - COPD
                - lung and pleural malignancy
                - infection
                    - pneumonia
                        - particularly pneumocystis jiroveci
                        - TB
                - suppurative lung disease
                    - cystic fibrosis
                    - bronchiectasis
                    - lung abscess
                - interstitial lung disease
                    - sarcoidosis
                    - idiopathic pulmonary fibrosis

        - hypersensitivity pneumonitis
        - pneumoconiosis
        - catamenial
            - 90% in the right lung
            - occurs up to 24 hours before or within 72 hours of the onset of menstruation
            - thoracic endometriosis leads to holes in the diaphragm which allow passage of air from the genital tract when the mucus plug is liquefied at the time of menstruation
            - ovarian suppression for 6-12 months is used to prevent recurrence
- clinical assessment
    - primary spontaneous pneumothorax
        - classic presentation is sudden onset of pleuritic chest pain and dyspnoea at rest
        - symptoms do not correlate with size of pneumothorax
        - many cases are mild and most patients will present after 2 days of symptoms
    - secondary spontaneous pneumothorax
        - symptoms tend to be more severe as underlying lung function is already compromised
        - primary complaint is breathlessness out of proportion to the radiological size of the pneumothorax
    - signs
        - affected by size of pneumothorax – a small one may not be clinically detectable
        - features of larger pneumothoraces include:
            - tachycardia
            - tachypnoea
            - reduced breath sounds on the affected side
            - reduced chest expansion on the affected side as the patient splints the chest wall
            - hyper-resonance on the affected side
            - decreased tactile vocal fremitus on the affected side
- investigation
    - CXR
        - PA views most useful
            - tend to underestimate size due to being 2D image of 3D structure
        - measurement should be from the lung edge to the thoracic wall at the level of the hilum
            - <2cm is small
            - >2cm is large
        - 2cm approximates to the point at which up to half of the lung volume has been lost
        - sensitivity is 80-85% for identifying small pneumothoraces
        - potential pneumothorax mimics:

                - medial border of the scapula
                - outline of the oxygen reservoir bag or associated tubing
                - clothing
                - bedsheets
                - companion shadows (visible subcostal groove, usually ribs 1 and 2)
                - skin folds
                - post-pleurectomy scarring/suture material
            - repetition of films with potential artefacts removed or comparison to previous images can be useful
        - CT
            - gold standard for identifying pneumothorax
            - valuable when x-rays difficult to interpret or specific drain placement is required e.g. bullous long disease, loculated pneumothoraces, surgical emphysema
        - ultrasound
            - sensitivities up to 95% reported
            - user dependent
            - more accurate than AP CXR if done by an experienced user
            - CXR has advantage of identifying unexpected causes of pleuritic pain such as infection or malignancy
        - ABG
            - unlikely to change management plan
            - main use is when supplementary oxygen is given to patients with COPD and pneumothorax
- management
    - depends on size of pneumothorax, whether it is primary or secondary and whether the patient is symptomatic
    - patients who are symptomatic or admitted for observation should have supplementary high flow oxygen
        - a pneumothorax will resolve 4 times faster on high flow oxygen than on room air
        - fixed oxygen concentration should be used for COPD patients
    - Entonox is contraindicated
        - diffuses into air spaces and can convert an uncomplicated pneumothorax into a tension pneumothorax
    - minimal symptoms
        - small spontaneous pneumothorax and minimal symptoms
            - 80% will have no ongoing leak
            - discharge with written advice and organised follow up
        - resolution on room air is at around 2% of hemi-thorax volume per day
            - a 1cm pneumothorax would resolve in around 12 days
            - a 2cm pneumothorax could take 3-4 weeks to fully resolve
        - patients with spontaneous secondary pneumothoraces less than 1cm with minimal symptoms do not require ED drainage but should be admitted for oxygen and observation
            - these patients should never be discharged from the ED and should be observed for a minimum of 24 hours

- symptomatic patients
    - a primary pneumothorax >2cm or symptomatic should be managed with needle aspiration (up to 2 litres)
        - they can be discharged with OP follow up in 3-4 weeks if symptoms improve and volume reduced to <2cm
        - if unsuccessful, they require chest drain and admission
    - a secondary pneumothorax of 1-2cm should be aspirated (up to 2.5 litres)
        - if successful (now <1cm) they should be admitted, otherwise a chest drain should be inserted
    - if >2cm or breathless, a chest drain should be inserted and the patient admitted
    - if <2cm and not symptomatic, they should be given oxygen and admitted for observation

- needle aspiration
    - initial success rate no difference to chest drain insertion for primary spontaneous pneumothoraces (60-70%)
    - successful aspiration is associated with a much higher rate of discharge and fewer complications
    - patients should have a short period of observation in the ED before discharge
    - recurrence rates are similar to chest drain insertion
    - BTS guidelines recommend use of a cannula no greater than 16G
        - evidence is limited for larger cannulae being more likely to cause persistent pleural leaks
        - narrow cannulae are also shorter and may not reach the pleural cavity in larger patients
    - less likely to succeed in secondary pneumothoraces
        - only recommended for small (1-2cm) pneumothoraces with minimal symptoms
    - procedure:
        - inform patient and obtain written consent
        - position patient in slightly reclined position
        - identify insertion point
            - mid clavicular line, second intercostal space
                - found by locating end of second rib medially where it attaches at the manubriosternal angle – the ribspace below is the 2nd intercostal space
            - mark with a pen
        - put on gown and sterile gloves, clean area with anti-septic solution and apply drapes
        - infiltrate a few ml of 1% lidocaine subcutaneously
        - insert 14G cannula attached to 10ml syringe containing normal saline in horizontal plane at insertion point
        - aspiration of air confirms correct placement
        - remove trochar
        - attach cannula to short piece of connecting tubing and 3-way tap
        - aspirate via 50ml syringe, turn tap and dispel air into the atmosphere
        - turn tap again, aspirate another 50ml and repeat

        - continue until patient coughs or 2.5 litres aspirated
        - repeat CXR
        - repeat aspiration if indicated
        - if successful, remove cannula and place small occlusive dressing over insertion site
        - if unsuccessful, remove cannula and insert thoracostomy tube
    - considerations
        - failure to attach the cannula to a connecting piece increases the likelihood of tube kinking or accidental removal
        - minimum of two people required – one to manually secure the cannula and turn the connector, the other to perform the aspiration
        - time consuming (50x 50ml) but success rates are relatively high
        - beware turning the tap the wrong way and inserting air into the pleural cavity
        - syringe gets warm with friction and increasing effort is required with each aspiration
- intercostal chest drain (thoracostomy tube)
    - small drains are associated with fewer complications and do not prolong time to resolution
    - Seldinger technique may not be possible in obese patients as needle may be to short to traverse chest wall
    - insertion should be in the safe triangle to avoid injury to the long thoracic nerve and lateral thoracic artery which are in the mid axillary line
    - recognised complications include:
        - failure
        - false passage (subcutaneous)
        - intra-peritoneal placement with liver or splenic injury
        - surgical emphysema
        - empyema
        - haemothorax (intercostal or lateral thoracic artery injury)
        - intercostal nerve injury
        - long thoracic nerve injury
        - lung laceration
        - tube blockage
        - myocardial injury
    - procedure
        - inform the patient of the procedure and obtain written consent
        - position the patient reclined at around 45 degrees with hand behind head
        - identify the insertion point – just anterior to the mid-axillary line, 5th intercostal space and mark with a pen
        - put on gown and sterile gloves, clean the area with antiseptic solution and apply drapes
        - using an aseptic technique, infiltrate 1% lidocaine (max 0.3ml/kg) subcutaneously and down to pleura
        - insert needle from the kit, attached to the provided syringe, slowly through the chest wall, just above the underlying rib, in a horizontal plane
        - stop advancing when aspiration of air confirms correct placement
        - thread the guidewire through the needle

        - remove the aspiration needle
        - dilate the track with the dilators – may require a nick in the skin with a scalpel
            - holding the dilators close to the chest wall should prevent excessive force of exertion or sudden give
        - feed the drain over the guidewire – usually up to 12cm in adults
        - remove guidewire and tube obturator
        - fogging of the tube suggests correct placement
        - connect the tube to an underwater drainage system below the level of the patient – an assistant should hold the drain to prevent it coming out before suturing
        - bubbling on coughing and a fluid swing confirm placement
        - secure with a stay suture
        - place a clear dressing over the insertion site
        - secure the drain to the chest wall with an omental tag
        - repeat the CXR
- prognosis and follow up
    - any patient requiring admission should be reviewed by a respiratory physician within 24 hours
    - drain suction
        - routine use is not recommended
            - may precipitate re-expansion pulmonary oedema
        - should be considered if re-expansion has not occurred after 48 hours – high volume, low pressure suction
- referral to cardiothoracic surgeon
    - referral criteria:
        - second ipsilateral pneumothorax
        - first contralateral pneumothorax
        - bilateral spontaneous pneumothorax
        - persistent air leak or failure of lung re-expansion 5 days after chest drain insertion
        - spontaneous haemothorax
        - professions at risk (e.g. pilots, divers)
        - pregnancy
            - risk of recurrence increases in pregnancy and poses risk to mother and foetus
        - surgical options include:
            - open thoracotomy, resection of pleural blebs and pleurectomy
                - gold standard at preventing recurrences
                - failure rate <1%
            - open thoracotomy, resection of pleural blebs and pleural abrasion/pleurodesis
                - recurrence rate approximately 2%
            - video assisted thorascopic surgery (VATS)
                - increasing in popularity due to reduced hospital stay and less postoperative pain

        - recurrence rate >5% - not recommended in people who wish to dive
    - chemical pleurodesis is an option for patients that refuse or are not candidates for surgery
        - administration of tetracycline or talc via chest drain
        - failure rate approximately 10%
- follow up
    - patients discharged from the ED after spontaneous pneumothorax should ideally be reviewed by a respiratory physician after 2 weeks
        - in practice it may be impossible to access clinics within this timeframe and the patient should be advised to return to the ED for repeat CXR and senior doctor review at 2 weeks
        - if pneumothorax is recurrent or profession high risk cardiothoracic referral is appropriate
    - advice
        - verbal and written advice to return immediately to the ED if they develop breathlessness
        - smokers should be advised to quit and seek help from the GP to achieve this
            - risk of pneumothorax in men who smoke >20 cigarettes/day is 100 times greater than in men who do not smoke
        - avoid flying for at least a week after CXR has confirmed resolution of spontaneous pneumothorax or until they have recovered from a definitive surgical procedure aimed at preventing recurrence
            - if pneumothorax was traumatic, 2 weeks should elapse following resolution before flying
            - changes in atmospheric pressure when flying can quickly convert simple pneumothoraces to tension pneumothoraces
        - diving should be permanently avoided unless the patient has had bilateral open surgical pleurectomy

RC9 pulmonary aspiration

- aspiration pneumonia
    - background
        - aspiration of oropharyngeal contents which includes colonising flora and leads to infection
        - elderly have increased colonisation with Staph aureus, aerobic Gram-negative bacilli (Klebsiella and E. coli) plus dysphagia which leads to pneumonia
        - distinguishing from aspiration pneumonitis is difficult and the distinction is controversial
    - risk factors
        - patient factors
            - full stomach
            - opioid use
            - pain
            - pregnancy
            - increased BMI

                - distended abdomen
                    - mass, ascites, bowel obstruction
                - GORD
                - decreased level of consciousness
                - gastric mass
                - diabetes
                - oesophageal motility disorders
                - anxiety
                - chronic neurological conditions (e.g. CVA, MS, MG, myotonic dystrophy)
                - scleroderma
            - anaesthetic factors
                - anaesthesia with unprotected airway
                - instrumentation of airway with inadequate depth of anaesthesia
                - paralysis
                - head down positioning
                - inadequate cricoid pressure
                - use of airways other than cuffed ETT
                - opioids
                - use of inhalational agent (N20)
            - surgical factors
                - laparoscopic insufflation of abdomen
                - bowel or visceral manipulation
                - pain
    - clinical features
        - generally not witnessed
        - typical symptoms
        - infiltrate in bronchopulmonary segment
        - posterior segments of upper lobes involved if patient aspirates in recumbent position
        - basal segments of lower lobes involved if patient upright
        - higher chance of cavitation and lung abscess formation
    - management
        - antibiotics, usually 7 days in total
- aspiration pneumonitis
    - background
        - also known as Mendelson syndrome
        - chemically induced inflammation of the lungs as a result of aspiration of gastric contents
            - chemical burn of the tracheobronchial tree and pulmonary parenchyma
                - causes an intense inflammatory reaction
    - pathophysiology
        - aspiration more likely with:
            - increased intragastric pressure
            - decreased upper and lower oesophageal sphincter tone
            - obtunded upper airway reflexes

        - factors reducing lower oesophageal pressure include
            - gastric fluid components
            - increased acidity
            - lipids
            - hyperosmolar fluid
            - progesterone
            - pharmacological agents
            - dopaminergic agonists
            - beta adrenergic agonists
            - theophylline and caffeine
            - anticholinergics
            - opioids
        - a SIRS response can occur in the absence of infection with raised WCC, fever, tachycardia and hypotension
- management of aspiration
    - immediate
        - minimise further aspiration
        - if awake, suction and place in recovery position
        - if breathing spontaneously – recovery position
        - if unconscious and apnoeic:
            - secure airway with cuffed ETT
            - suction until airway clear
            - 100% O2
            - CPAP
    - subsequent
        - empty stomach with NG tube
        - CXR – diffuse infiltration, often in RLL
        - bronchoscopy +/- lavage
        - chest physiotherapy
        - ITU referral if appropriate
        - antibiotics not indicated unless aspiration of infected material is a particular concern or patient has other risk factors

RC10 pulmonary embolus

- background
    - annual incidence 60-70/100,000
        - half of cases occur in hospital or long term care settings
    - around 25,000 patients in the UK die every year from potentially preventable hospital acquired VTE
    - the majority of fatal PEs are not clinically suspected prior to death
    - only around 10% of patients with suggestive symptoms are found to have a PE
    - treatment leads to around a 20% reduction in mortality
- pathophysiology
    - PE is a complication of underlying VTE
    - normally, tiny microthrombi are continually formed and lysed within the venous circulatory system

        - in some pathological situations, microthrombi escape the fibrinolytic system and are able to propagate into larger thrombi
    - predisposing factors are based on Virchow's triad
        - venostasis
            - immobility
            - paralysis
            - atrial fibrillation
            - congestive heart failure
        - hypercoagulability
            - malignancy
            - pregnancy
            - protein C and S deficiency
            - antithrombin deficiency
        - vessel wall inflammation
            - indwelling catheters
            - trauma
            - surgery
    - most PEs occur when fragments of a thrombus break free from the deep venous system, typically in the pelvis or lower limb, up through the vena cava, through the right atrium and into the pulmonary circulation
        - up to 80% of patients with PE have detectable clots in the leg or thigh
        - clots can form in the upper limbs but are less likely to embolise and tend to cause smaller, less clinically significant emboli when they do
    - the lungs act as a filter for blood returning to the heart, so all but the smallest clots get stuck there rather than travelling into the arterial circulation
        - lungs have a dual blood supply from the pulmonary artery and bronchial arteries which helps them recover function after small pulmonary emboli
- types of PE
    - provoked
        - associated with a transient risk factor such as significant immobility, surgery, trauma, pregnancy, puerperium
    - unprovoked
        - occurs in the absence of a transient risk factor
        - person may have no identifiable risk factor or a risk factor that is not easily correctible
    - massive PE
        - more than 50% obstruction of the vascular bed
        - most immediate problem is usually circulatory collapse due to fall in cardiac output as the right heart fails to pump an adequate volume of blood through the reduced pulmonary vasculature to the left side of the heart
        - in younger patients with good pulmonary function, high flow oxygen may be sufficient to correct hypoxia as all of the blood reaching the left heart is passing through good quality lung tissue giving good oxygenation
        - a non-massive PE may present significant problems in patients with severe underlying lung disease
- clinical assessment
    - massive PE

        - uncommon
        - patient is clinically shocked and hypoxic
        - clinical diagnosis is usually straightforward
            - other conditions such as right ventricular infarction, sepsis and concealed upper GI haemorrhage can present in a similar fashion
        - risk of death within 90 days is about 50%
    - submassive PE
        - <50% obstruction of pulmonary vascular bed
        - patient has acute dyspnoea and frequently chest tightness/pain
        - differential includes acute coronary syndromes, pneumonia and heart failure
    - pulmonary infarction
        - commonly presents with pleuritic chest pain which may be associated with haemoptysis (uncommon) and/or dyspnoea
        - usually the group with the most diagnostic uncertainty
        - differentials include musculoskeletal pain, pleurisy, viral and bacterial infections, pericarditis, pneumothorax
        - clinical examination should focus on signs of deep vein thrombosis and conditions that may masquerade as PE
            - chest examination is usually normal in PE
            - signs of underlying causes of PE should also be sought, e.g. organomegaly, lymphadenopathy
- clinical features
    - in descending order of frequency:
        - dyspnoea (70%)
        - tachypnoea (RR >20)
        - pleuritic chest pain
        - apprehension
        - tachycardia (>100bpm)
        - cough
        - haemoptysis
        - leg pain
        - clinically evident DVT (10%)
    - less than 10% of patients with PE have none of the top 3 symptoms
    - none of the symptoms are pathognomic
    - some patients present with more subtle symptoms or atypical symptoms such as syncope or postural hypotension
    - diagnosis is more difficult in subacute PE as the patient may complain of gradually increasing dyspnoea or reduced exercise tolerance rather than acute symptoms
- risk factors
    - major (relative risk 5-20)
        - surgery
            - major abdominal/pelvic surgery
            - hip or knee replacement
            - post-operative intensive care
        - obstetrics
            - late pregnancy

                - caesarean section
                - puerperium
            - lower limb problems
                - fracture
                - varicose veins
            - malignancy
                - abdominal/pelvic
                - advanced/metastatic
            - reduced mobility
                - hospitalisation
                - institutional care
            - previous proven venous thromboembolism
        - minor (relative risk 2-3)
            - cardiovascular
                - congenital heart disease
                - congestive cardiac failure
                - hypertension
                - superficial venous thrombosis
                - indwelling central vein catheter
            - oestrogens
                - oral contraceptive
                - hormone replacement therapy
            - miscellaneous
                - COPD
                - neurological disability
                - occult malignancy
                - thrombotic disorders
                - long distance sedentary travel
                - obesity
- investigations
    - CXR
        - usually normal
        - may show changes such as:
            - small pleural effusion
            - wedge shaped pulmonary infarct
            - regional oligaemia (Westermark sign)
        - used to diagnose or exclude other possible conditions
    - ECG
        - signs supportive of right heart strain such as right axis deviation
        - rule out acute coronary syndrome
        - most common finding in PE is sinus tachycardia
        - atrial fibrillation present in 20% of cases
        - S1 Q3 T3 pattern reflects right heart strain and is seen in only 10%
    - ABG
        - may confirm hypoxia, hyperventilation and increased Arterial-alveolar gradient
            - PAO2-PaO2

                - PAO2 = PiO2-PaCO2/0.8
            - normal A-a gradient for a young adult non-smoker breathing air is 5-10mmHg
                - gradient increases with age
        - features may also be found in other conditions such as pneumonia
    - FBC
        - marked neutrophilia may suggest infection rather than infarction
    - troponin
        - may be elevated in acute PE, particularly massive PE
        - high levels suggest right ventricular strain or overload
            - can be helpful for prognosis or risk stratification (e.g. decision making for thrombolysis)
            - not helpful for diagnosis of PE
- risk stratification
    - most commonly used tools are:
        - Well's criteria
        - BTS risk stratification (superseded by NICE guidance in the UK)
        - Revised Geneva Score or Simplified Revised Geneva Score
        - Pulmonary Embolus Rule-Out Criteria (PERC)
    - Well's score
        - semi-objective way of calculating pre-test probability
        - criteria:
            - clinically suspected DVT (3.0)
            - PE at least as likely or more likely than alternative diagnosis (3.0)
            - pulse rate >100 (1.5)
            - immobilisation >3 days (1.5)
            - surgery in last 4 weeks (1.5)
            - previous VTE (1.5)
            - haemoptysis (1.0)
            - malignancy (1.0)
        - patients scoring 4 or less are low risk (PE unlikely)
        - patients scoring more than 4 are high risk (PE likely)
    - BTS Guideline 2003
        - question 1: is a major risk factor for PE present?
        - question 2: is another reasonable clinical explanation for the symptoms unlikely or absent
            - high risk: answer to both questions is yes
            - intermediate risk: answer to one question is yes
            - low risk: answer to both questions is no
    - Revised and Simplified Revised Geneva Score
        - does not rely on clinical gestalt
        - widely used in Europe
        - similar performance to Well's scoring
        - criteria
            - age >=65
                - revised: 1
                - simplified: 1

- previous DVT/PE
    - revised: 3
    - simplified: 1
- recent surgery/fracture (<4 weeks)
    - revised: 2
    - simplified: 1
- active malignancy
    - revised: 2
    - simplified:1
- unilateral lower limb pain
    - revised: 3
    - simplified: 1
- haemoptysis
    - revised: 2
    - simplified: 1
- pulse 75-94bpm
    - revised: 3
    - simplified: 1
- pulse >=95
    - revised: 5
    - simplified: 2
- pain on lower limb deep vein palpation or unilateral oedema
    - revised: 4
    - simplified: 1

▪ revised score:
- 0-3: low risk
- 4-10: moderate risk
- 11-25: high risk

▪ simplified revised score:
- 0-2: low risk
- 3 or more: high risk

o PERC

▪ allows re-stratification of some patients into a no risk group where no further investigations are required
- they have a less than 2% chance of having a PE

▪ criteria:
- low risk by gestalt or other criteria
- age <50
- pulse <100
- oxygen saturations on room air >94%
- no unilateral leg swelling
- no haemoptysis
- no recent trauma or surgery
- no previous VTE
- no oral hormone use

▪ all answers must be yes

- further investigations

- d-dimer
  - sensitive but not specific so has poor predictive value
  - also elevated in infection, trauma, cancer and inflammatory conditions
  - should only be used in patients in whom a negative result will reliably exclude PE (low risk patients)
  - a negative result in a high risk patient does not exclude PE
  - there are two types of assay:
    - qualitative bedside assays
    - quantitative laboratory based assays
- radiological investigations
  - for low risk patients with positive d-dimer or for high risk patients
  - techniques include:
    - CT pulmonary angiogram
    - V/Q scanning
    - echocardiography (particularly in patients too sick to be moved from the ED)
    - leg vein ultrasound (limited applicability)
  - CTPA
    - investigation of choice
    - greater sensitivity and specificity for PE then V/Q scan and has the ability to identify alternative diagnoses
    - sensitivity is 85-90%
    - negative predictive value is high (96-97%)
    - there is no evidence available to suggest that some of the clots found may not require treatment
    - CTPA may be falsely negative in patients who are high risk for PE and further investigation such as leg ultrasound, MRI or invasive pulmonary angiography may be required
  - isotope lung scanning (V/Q scan)
    - can be used for patients with a normal CXR and no chronic cardiopulmonary disease
    - risk stratifies into low, intermediate and high probability of PE
      - for low risk patients with a low probability V/Q scan, PE is considered to have been excluded
      - for high risk patients with a high probability V/Q scan, PE is considered confirmed
    - for many patients, the scan does not provide a definitive diagnosis
  - echo
    - may provide indirect evidence of PE with right ventricular strain and high PA pressures
      - clot in the right side of the heart or pulmonary artery is only rarely seen
  - ultrasound
    - identification of leg DVT in symptomatic patients can preclude the need for further imaging (e.g. in pregnancy)
    - negative scan does not exclude subclinical DVT

- leg ultrasound alone cannot reliably exclude VTE in patients presenting with signs and symptoms of PE
- it should be considered in patients in the high risk PE group with negative CTPA, particularly if they also complain of unilateral leg pain or swelling

- management
  - patients awaiting investigation
    - patients in the PE likely subgroup and the PE unlikely subgroup with positive d-dimer should have anticoagulation (usually LMWH) whilst awaiting further investigation
    - anticoagulation should only be delayed if CTPA is immediately available
  - stable patients with confirmed PE
    - oxygen
      - if sats <94% on room air
    - anticoagulation
      - no clear evidence that 6 months is better than 3 months, but bleeding risk is increased
      - decision to continue beyond 3 months needs to be based on individual's risk of recurrence vs bleeding risk
      - with multiple episodes or continuing risk factors such as malignancy, lifelong anticoagulation should be recommended
      - initial management is with LMWH
        - patients can then be started on DOAC or warfarin according to local guidelines
  - unstable patients with suspected or confirmed PE
    - thrombolysis
      - indicated for patients with severe circulatory compromise or a picture of massive PE
      - prior proof of PE not required if patient is peri-arrest
        - 50mg alteplase as a bolus should be administered immediately
        - in the setting of massive PE, only active internal bleeding or recent intracranial bleed are absolute contraindications
      - patients who are too unstable for investigation can be considered for thrombolysis as either bolus or infusion
      - in patients with non-massive PE there is no benefit from routine thrombolysis as outcome is good anyway
  - outpatient management
    - the simplified Pulmonary Embolism Severity Index (PESI) can be used to identify patients suitable for outpatient/ambulatory care
      - age >80 (+1)
      - history of cancer (+1)
      - history of chronic cardiopulmonary disease (+1)
      - heart rate >= 110 (+1)
      - systolic BP <100 (+1)
      - O2 saturation <90%) (+1)
    - >0 points is high risk

- PE in special circumstances
    - active cancer
        - LMWH heparin may be a better choice than warfarin
            - better reduction in recurrence of VTE
            - INR control is more difficult in patients receiving chemotherapy
            - patients with active cancer are at greater risk of significant bleeding as a result of anticoagulation
        - a discussion should be had over risks/benefits of continuing after 6 months – current recommendation is to continue with anticoagulation lifelong in patients with active cancer
    - pregnancy
        - d-dimer levels are often raised in pregnancy, limiting its use as a triage tool
        - bilateral leg vein ultrasonography may be used as a first step
        - total radiation doses with CTPA and V/Q scans are small
            - CTPA gives a larger dose of radiation to maternal breast tissue
            - V/Q gives a larger dose of radiation to the foetus
                - slightly increased risk of childhood cancer compared with CTPA (1 in 280,000 vs 1 in 1,000,000)
                - lower risk of maternal breast cancer
                    - lifetime risk increased by up to 13.6% with CTPA
        - LMWH is the treatment of choice for PE in pregnancy
            - twice daily regime indicated due to different pharmacokinetics
                - 1mg/kg enoxaparin BD
        - oral anticoagulation not used due to greater bleeding risk and teratogenic risk
        - temporary IVC filter may be fitted prior to delivery as anticoagulation will need to be stopped due to risk of haemorrhage
    - intravenous drug users
        - mechanism probably related to recurrent femoral vein puncture with injection of irritant substances
        - there is limited evidence as to the risk of developing PE secondary to VTE in this group
        - PE is often associated with infection and septic emboli can occur
        - foreign particle embolization can also occur with needles, talc, cotton etc
            - this can cause granuloma formation and subsequent deterioration in lung function
        - management can be difficult due to chaotic lifestyles
        - continuation of injection into large veins can pose bleeding risk
        - drug users are often managed with LMWH for 3-6 months
        - antibiotics should be considered in addition
        - IVC filters can be considered if long term anticoagulation is inappropriate

RC11 viral induced wheeze in children

- background
    - generally affects children between the ages of 6 months and 5 years
    - wheeze is associated with respiratory tract infection
    - mechanism is poorly understood, but these children have smaller airways

    - respiratory tract infection is associated with at least one episode of wheeze in around 50% of children before 6 years of age
    - around 15% of children with viral wheeze have asthma and continue to wheeze after 5 years of age
- clinical features
    - under 5 years
    - respiratory rate normal or increased
    - hyperinflation may be present
    - wheeze is present
    - crackles are not usually present
    - onset is usually acute
- management
    - mild
        - 6-10 puffs of salbutamol via spacer
        - if good response, child may be discharged with an inhaler and spacer and an information leaflet
        - if no response, escalate treatment and review diagnosis
    - moderate/no response to initial treatment
        - 'hour of power' – 10 puffs of salbutamol via spacer repeated at 15-30 minute intervals until 3x10 puffs given
        - observe and reassess throughout
        - good response may lead to discharge
        - moderate response may need more time with hourly inhalers
        - if deterioration or no response, escalate treatment and review diagnosis
    - severe/not responding to treatment
        - get help, including crash team if needed
        - oxygen
        - continuous nebulised bronchodilators as per BTS guideline
        - if deteriorating or failing to respond, start intravenous therapy as per guideline (salbutamol, aminophylline, magnesium, hydrocortisone)
    - CXR should be avoided unless it is a severe episode or there is a specific indication
    - steroids are given for severe episodes but there is no evidence that they help if given routinely
    - patients with increased work of breathing and a history of viral wheeze who have a clear chest may still settle with salbutamol
        - they may develop wheeze as bronchospasm eases
        - other diagnoses should also be considered in atypical presentations

SEXUAL HEALTH

SeP1 genital discharge

- vaginal discharge
    - background
        - normal discharge is a white or clear non-offensive discharge that varies with the menstrual cycle
    - causes

        - non-infective
            - physiological
                - newborn infants may have some discharge, sometimes mixed with blood due to high levels of circulating maternal oestrogen
                    - should disappear by 2 weeks of age
                - during the reproductive years there is a change in discharge due to fluctuating levels of oestrogen and progesterone
                    - when oestrogen is low, the mucus is thick and sticky; as levels rise the mucus becomes clearer, wetter and more stretchy; after ovulation, it becomes increasingly thick and sticky again
                - at menopause, the normal amount of discharge decreases as oestrogen levels fall
            - cervical polyps and ectopy
            - foreign bodies – e.g. retained tampon
            - vulval dermatitis
            - erosive lichen planus
            - genital tract malignancy, e.g. cancer if the cervix, uterus or ovary
            - fistulae
        - non-sexually transmitted infection
            - bacterial vaginosis
                - most commonly seen in sexually active women
            - candida infections
        - sexually transmitted infections
            - Chlamydia trachomatis
            - Neisseria gonorrhoeae
            - Trichomonas vaginalis
                - frequently found in association with infection with N. gonorrhoeae
    - assessment
        - full clinical and sexual history
        - nature of discharge
            - what has changed
            - odour
            - onset
            - duration
            - colour
            - consistency
        - associated symptoms
            - itch
            - superficial dyspareunia or dysuria
            - abdominal pain
            - deep dyspareunia
            - abnormal bleeding
            - pyrexia
        - assessment of risk of STI

- increased in:
    - women under 25
    - women who have a new sexual partner
    - women who have had more than one partner in the preceding 12 months
- ask when she last had sex and when she last had sex with anyone else

- current contraceptive use
- concurrent medications (e.g. antibiotics, steroids)
- previous treatments used (prescription and over the counter)
- medical conditions (e.g. diabetes, immunocompromise)
- symptoms suggesting discharge is abnormal include:
    - heavier than usual discharge
    - thicker than usual discharge
    - pus-like discharge
    - white and clumpy discharge
    - greyish, greenish, yellowish or blood-tinged discharge
    - foul smelling (fishy or rotting meat) discharge
    - discharge accompanied by bloodiness, itching, burning, rash or soreness

- presentation
    - infective (non-sexually transmitted) vaginal discharge
        - BV
            - thin, profuse, fishy-smelling discharge without itch or soreness
        - candidiasis
            - thick, typically curd-like, white, non-offensive discharge associated with vulval itch and soreness
            - may cause mild dyspareunia and dysuria
    - infective (STI) vaginal discharge
        - C. trachomatis, N. gonorrhoeae and T. vaginalis can present with discharge but may be asymptomatic – they are associated with increased risk of HIV transmission
        - C. trachomatis
            - may cause copious purulent vaginal discharge
            - asymptomatic in 80%
        - T. vaginalis
            - may cause an offensive yellow vaginal discharge, often profuse and frothy, associated with vulval itch and soreness, dysuria, abdominal pain and superficial dyspareunia
        - N. gonorrhoeae
            - may present with a purulent vaginal discharge
            - asymptomatic in up to 50%
    - non-infective causes
        - retained foreign bodies
            - foul-smelling serosanguinous discharge
            - diagnosis confirmed on examination

- cervical polyps and ectopy
    - usually asymptomatic
    - may have increased discharge and intermenstrual bleeding
    - diagnosis made on speculum examination
- genital tract malignancy
    - presentation varies
    - persistent discharge not responding to conventional treatment may be the first clue
    - diagnosis made on examination and biopsy
- fistulae
    - history of trauma or surgery is suggestive
    - may be foul or feculent discharge in association with recurrent UTIs
- allergic reactions
    - diagnosis suspected on taking history – e.g. use of irritant chemicals in douching, contact with latex or semen
- vulval lichen planus and vulval dermatitis are apparent on examination but may require biopsy to confirm diagnosis

- investigations
    - patients with typical symptoms of BV or vulvovaginal candidiasis at low risk of STI can be treated without sampling
    - otherwise, STI screening and swabs should be offered:
        - endocervical swab in transport medium for gonorrhoea
        - endocervical swab for chlamydial nucleic acid amplification test (NAAT)
        - if examination is declined, a self-taken vulvovaginal swab may be an option
        - blood tests for HIV and syphilis
    - vaginal pH testing can help differentiate between BV (which reduces normal vaginal acidity to >4.5) and vulvovaginal candidiasis (pH <4.5)
    - high vaginal swab is only useful if there are recurrent symptoms, treatment failure, pregnancy, postpartum, post-abortion, post-instrumentation
- management
    - referral to GUM service for STI or recurrent infections
    - finding of STI should prompt patient education and counselling, screening for other STIs and contact tracing for appropriate testing and management
    - infective, non-sexually transmitted causes
        - BV
            - metronidazole and clindamycin either orally or vaginally
            - testing and treatment of male partners not indicated but should be considered for female sexual partners
        - vaginal candidiasis
            - vaginal or oral azole antifungals
            - oral treatment should be avoided in pregnancy
            - topical antifungals may be used in addition where there are vulval symptoms

                - no need for routine screening or treatment of sexual partners
                - latex contraceptives may be weakened by antifungal treatments
        - non-infectious causes
            - retained foreign bodies
                - manual removal
                    - very small pieces may not be visible and may require a lavage (may require sedation in children)
                - sedation or anaesthesia may be required for larger or irregularly shaped objects causing vaginal wall spasm
                - short course of antibiotics may be required if the object was there for long enough to cause secondary infection
            - cervical polyps can be removed and sent for histology
            - cervical ectopy can be managed by a gynaecologist
    - prevention
        - basic personal hygiene (without the use of douches or perfumed chemical agents) with avoidance of tight synthetic clothing
        - emollients can be used as a soap substitute and moisturiser to prevent dryness and maintain the skin's natural barrier function
        - treatment of sexual partners as appropriate
- male urethritis
    - background
        - urethral inflammation which can be the result of infectious or non-infectious causes but is primarily sexually acquired
    - features
        - mucopurulent or purulent discharge from the urethral meatus
        - Gram stain of urethral smear shows >5 polymorphonuclear (PMN) cells per high power field
        - first pass urine positive for >10 PMN per high power field
            - sensitivity is low for positive leucocytes
    - classification
        - gonococcal urethritis
            - causes by Neisseria gonorrhoeae
        - non-gonococcal urethritis
            - caused by other agents including non-infective agents
            - most commonly Chlamydia trachomatis and Mycoplasma genitalium
            - neither detected in 30-80%
                - pathogen-negative urethritis is more likely with increasing age and in the absence of discharge or clinical symptoms
            - Trichomonas vaginalis is more common in non-white ethnic groups
            - ureaplasmas may account for some cases
            - other possible causes include urinary tract infections, adenovirus, herpes simplex, EBV, candida, urethral stricture, foreign body
        - persistent or recurrent urethritis
            - 10-20% of cases treated for non-gonococcal urethritis
            - probably multifactorial but usually no identifiable cause

- epidemiology
  - most common condition diagnosed and treated in men attending GUM clinics in the UK
  - >80,000 cases/year
- presentation
  - may be asymptomatic
    - 90-95% of men with gonorrhoea
    - 50% of men with chlamydia
  - urethral discharge
    - mucopurulent or purulent
    - with or without blood
    - more noticeable after holding urine overnight
    - more common in gonococcal infection
    - may be unnoticed by patient and only seen on examination
  - urethral pruritus, dysuria or penile discomfort with risk of sexually transmitted infection (sexually active and not used a condom or has recent new sexual partner)
  - other symptoms associated with the cause (e.g. skin lesions in herpes simplex)
  - systemic symptoms if involvement of other organs (e.g. conjunctivitis, arthritis)
  - examination may be normal or show haematuria and/or lymphadenopathy
- high risk patients
  - sexually active
  - male
  - unprotected vaginal sex
  - men who have sex with men or are bisexual
  - more common in cities
  - age <35-40 years
  - recent partner change
- investigations
  - should be at GUM clinic or primary care clinic with enhanced sexual health facilities
  - diagnosis is confirmed by demonstrating an excess of polymorphonuclear lymphocytes in the anterior urethra
    - usually with urethral smear, can be first pass urine specimen
  - pharyngeal and rectal swabs may also be needed
  - urine dipstick should be done to exclude UTI
  - patients should be counselled and offered testing for HIV, hepatitis B and syphilis
- differential
  - physiological discharge
  - candida balanitis
  - epididymo-orchitis
  - cystitis
  - acute prostatitis
  - urethral malignancy

- management
  - refer to GUM clinic
  - if specific test results available (follow local guidelines)
    - non-gonococcal urethritis
      - doxycycline 100mg BD for 7 days or azithromycin 1g stat
    - gonococcal urethritis
      - ceftriaxone 500mg IM stat plus azithromycin 1g PO stat
  - patient education
    - explanation of diagnosis, treatment, adverse effects and importance of completing course of antibiotics
    - methods of prevention including advice on safe sex
    - advise the patient to avoid sexual intercourse until infection has cleared and that partners should also be checked
    - contract tracing via GUM clinic
    - follow up review at 2 weeks with test of cure for gonorrhoea
- complications
  - epididymitis and/or orchitis
  - prostatitis
  - systemic dissemination of gonorrhoea – e.g. conjunctivitis, skin lesions
  - reactive arthritis
  - pelvic inflammatory disease in female partners
  - increased risk of HIV transmission

SeP2 genital lesions

- background
  - genital ulcers can be infectious or non-infectious in origin
- causes
  - infectious
    - genital herpes simplex virus (HSV)
      - HSV2 is mainly associated with genital infections but can be HSV1
      - resides in a latent state in the nerves that supply sensation to the skin
      - most people have no or mild symptoms and are not aware of being infected
        - found in around 1 in 5 adults
      - transmission
        - direct skin to skin contact
        - sexual, including oro-genital, contact is the most common method of spread
        - vertical transmission and self-inoculation can occur
      - primary genital infection
        - penile ulceration – painful, lasting 2-3 weeks if untreated with enlarged and tender local lymph nodes
        - pain and difficulty passing urine in females
        - may be systemic flu-like symptoms
      - recurrent genital herpes
        - triggers:

                - minor trauma
                - other infections
                - ultraviolet radiation (sun)
                - menstrual cycle
                - emotional stress
                - often no reason found
            - blisters are usually smaller in size and more closely grouped
                - these then produce shallow ulcers
            - shorter duration (5-10 days)
        - complications:
            - urethritis
            - proctitis
            - neurogenic pain
            - meningitis (rare)
            - widespread infection (debilitated or immunosuppressed patients)
        - pregnancy
            - minimal risk of transmission in recurrent infections
            - highest risk to baby when first episode occurs in the first few weeks or last few weeks of pregnancy
        - management
            - antivirals
                - may require repeated or continuous prophylactic courses
                - acyclovir or valciclovir PO
- syphilis
- chancroid
    - background
        - caused by Gram-negative bacterium Haemophilus ducreyi
        - incidence unknown but rare in most developed countries
        - likely to enter the host through microscopic breaks in the skin during sex
    - clinical features
        - onset typically 4-10 after sexual contact
        - one or more erythematous papules which evolve to pustules and become larger until they break down into an ulcer
            - typical ulcer is deep, with shaggy undetermined borders, and is very painful
            - base of ulcer has purulent exudate and bleeds easily
        - affects sites most prone to friction during sex – foreskin, glans and corona in men
        - if untreated, 50% develop infected lymph glands which become buboes on one or both sides of the groin around 1-2 weeks after initial ulcers
            - these can burst and discharge pus
        - in women, ulcers may be on the labia, vaginal entrance, cervix, perineum or perineal area

        - women generally have non-specific symptoms such as pain on urination or defecation, vaginal discharge, dyspareunia, rectal bleeding
    - untreated ulcers can persist for 103 months and result in scarring
    - they increase risk of HIV transmission
- diagnosis
    - made on probable diagnosis criteria:
        - presence of one or more painful genital ulcers
        - exclusion of syphilis
        - exclusion of herpes simplex infection
        - clinical presentation typical of chancroid (ulcers and lymphadenopathy)
    - culture of ulcer base material may be done but not reliable for diagnosis
- treatment
    - antibiotics (azithromycin, ciprofloxacin, ceftriaxone, erythromycin)
    - follow up to ensure resolution
    - drainage of fluctuant buboes may be needed
    - sexual health testing and contact notification

- lymphogranuloma venereum
    - background
        - caused by specific strains of Chlamydia trachomatis
        - outbreaks have occurred in men who have sex with men
        - transmitted by sexual penetration or skin to skin contact and sharing of equipment
    - clinical features
        - incubation period 10-14 days on average
        - primary infection
            - small painless genital papules, pustule or shallow ulcers
            - transient, often go unnoticed
        - secondary infection
            - 2-6 weeks after primary infection
            - painful and swollen lymph glands in the groin area
                - usually unilateral, can be bilateral
                - buboes can rupture and drain pus
            - women may have less specific symptoms such as pelvic and back pain
            - recipients of anal intercourse tend to present with:
                - anal/rectal pain
                - discharge
                - bleeding
                - tenesmus
                - constipation

        - other symptoms include malaise, fever, joint and muscular pains, vomiting
    - late stage
        - prolonged untreated infection may lead to complications such as:
            - abscesses
            - fistulas
            - lymphatic obstruction
            - severe genital oedema
            - rectal stricture
            - frozen pelvis
            - infertility
            - genital deformation
- diagnosis and management
    - laboratory testing as per microbiology advice
    - antibiotics (doxycycline or erythromycin)
    - buboes may require drainage
    - surgical management may be required (e.g. for fistula repair)

- granuloma inguinale (donovanosis)
    - caused by Klebsiella granulomatis
        - also called donovanosis after Donovan bodies, cellular components seen under the microscope
    - mostly found in tropical or subtropical areas
    - sexually transmitted, occasionally transmitted through skin to skin contact
    - ulcerovegetative type
        - single or multiple single or firm papules or nodules at the site of contact
        - these erode into soft, painless ulcers that bleed easily
        - ulcer base is beefy red granulation tissue with sharp margins and rolled or heaped up borders
        - continue to enlarge without treatment
    - nodular type
        - less common, mixture of papules and nodules with some granulation tissue
    - hypertrophic verrucous type
        - large vegetating masses, walnut like
    - necrotic type
        - deep foul smelling ulcer
    - cicatricial sclerotic type
        - extensive plaques of scar tissue
    - diagnosis is by direct microscopy of tissue
    - treated with antibiotics
- fungal infection (e.g. candida)
    - affects 75% of women at least once
    - overgrowth of vaginal candida

- C. albicans thrives on glycogen which is present in the vagina once oestrogen causes the lining to mature
- most common in women of reproductive age
- associated factors:
    - flares just before and after menstruation
    - pregnancy
    - high dose combined oral contraceptive pill
    - oestrogen based hormone replacement therapy after menopause, including vaginal cream
    - broad spectrum antibiotics
    - diabetes
    - obesity
    - iron deficiency anaemia
    - immunodeficiency
    - underlying skin conditions
    - other illness
- symptoms
    - itching, soreness and burning discomfort in the vagina and vulva
    - dysuria
    - vulval oedema, fissures and excoriations
    - dense white curd like discharge
    - bright red rash affecting inner and outer parts of vulva, sometimes spreading to groin and thighs
- symptoms may last hours, days, weeks or months
- diagnosis is clinical and can be confirmed by microscopy
- treatment is with topical antifungals
    - occasionally oral antifungals for persistent or recurrent episodes

▪ secondary bacterial infection

o non-infectious

▪ Behçet syndrome
▪ fixed drug eruption
▪ psoriasis
▪ sexual trauma
▪ granulomatosis with polyangiitis
▪ non sexually acquired genital ulceration
- painful ulceration of the external genitalia, usually in adolescents
- cause not fully understood
- follows an acute systemic illness (e.g. tonsillitis, URTI or diarrhoeal illness)
    - majority of cases associated with EBV
    - other associated causes include:
        ▪ CMV
        ▪ Mycoplasma pneumoniae
        ▪ Group A Strep
        ▪ Influenza A

        - salmonella
        - mumps
    - mainly affects the vulva of adolescent girls who are not sexually active
        - rarely can cause penoscrotal ulcers in young adult males
    - presentation
        - one or more well-defined, deep, punched out ulcers on the mucosal aspects and adjacent skin of the vulva or penoscrotal area
        - centre of ulcer is usually yellowish, may become black due to tissue necrosis
        - red rim around ulcer is usually at least 1cm in diameter
        - there may be mirror image 'kissing' lesions
        - there may be considerable swelling
        - typically very painful, result in dysuria or retention which may require admission and catheterisation
        - local lymph nodes may be enlarged and tender
    - diagnosis (both major and at least two minor criteria)
        - major criteria
            - acute onset of one or more painful vulval ulcers
            - exclusion of infectious and non-infectious causes of genital ulcers
        - minor criteria
            - ulcers of the vestibule or labium minorum
            - no history of sexual intercourse in the past 3 months or ever
            - flu-like symptoms
            - systemic illness in preceding 2-4 weeks
        - infectious mononucleosis testing is appropriate
    - management
        - reassurance and symptomatic treatment
            - analgesia – paracetamol, NSAIDs or stronger
            - short term catheterisation if pain inhibiting urination
            - topical anaesthetic ointment or gel
            - local hygiene measures and wound care
        - topical or oral steroid are sometimes used in severe cases but evidence is lacking
    - prognosis
        - usually resolves in an average of 15 days without scarring
        - rarely recurs

SeP3 emergency contraception

- background
    - 3 forms currently available in the UK
        - oral progestogen only emergency contraceptive (POEC) – levonorgestrel
        - selective progesterone receptor modulator (SPRM) – ulipristal acetate

        - copper intra-uterine contraceptive device (Cu-IUCD)
    - it is not considered an abortifactant
    - there is no time in the menstrual cycle when there is no risk of pregnancy, particularly in an irregular cycle
    - a Cu-ICD is the most effective form of emergency contraception and should be offered to all women
        - it can be inserted up to five days after the first unprotected sexual intercourse in a natural menstrual cycle or up to five days after the earliest likely date of ovulation (whichever is later)
    - evidence suggests that oral emergency contraceptives administered after ovulation are ineffective
- indications
    - when no contraception has been used
        - following consensual sexual intercourse
        - following rape or sexual assault
    - when there is contraceptive failure or incorrect use
        - incorrect use or failure of barrier methods such as condom, diaphragm or cap
        - failed coitus interruptus
        - miscalculation of the fertile time or failure to abstain/withdraw/use barrier methods during this time when using natural family planning methods
        - IUCD/intrauterine system expulsion
            - complete expulsion or the need to remove it midcycle and there has been unprotected sex within the last 5 days
        - whilst using any form of oral hormonal contraceptive if unprotected sexual intercourse occurs whilst taking or within 28 days of taking enzyme-inducing agents such as rifampicin
            - the Cu-ICD should be offered, or a double dose of LNG
        - incorrect use or potential failure of hormonal method of contraception
            - combined oral contraception
                - two or more pills missed in first seven days of the packet and unprotected intercourse has taken place in this period or the 7 day pill free period
                - if no extra barrier method used during episode of vomiting or severe diarrhoea lasting more than 24 hours
                - additional barrier methods are required for 7 days (LNG) or 14 days (UPA) whilst continuing normal pill taking
                - if more than 2 pills are missed during the last 7 days of the packet, the next packet should be started immediately without a pill free period
            - progestogen only contraception
                - if one or more pills has been missed or taken more than 3 hours late and UPSI has occurred in the 2-3 days prior to the missed pill or before two further tablets have been correctly taken
                - additional barrier methods are required for 2 days (LNG) or 9 days (UPA)

- medroxyprogesterone acetate (Depo-Provera)
    - if UPSI has taken place >14 weeks after the last injection
- contraceptive patches
    - if application of a new patch at the start of a cycle is delayed by more than 48 hours and UPSI has occurred – a new day 1 patch should be applied as soon as remembered
    - if the patch lifts partially or completely for more than 48 hours or if there is a delay of more than 48 hours in changing patches at the end of week 1 or week 2
    - additional barrier methods for 7 days (LNG) or 14 days (UPA)
- combined contraceptive vaginal ring
    - ring expelled for more than 3 hours during the first or second week of use and UPSI or barrier failure occurs in the 7 days following this
    - ring is found to be broken during use and UPSI has occurred in the previous 5 days or UPSI/barrier failure occurs in the seven days following this
    - ring is not immediately replaced after the 7 day ring free break and UPSI occurs during the ring free interval
    - additional barrier use for 7 days (LNG) or 14 days (UPA)
- contraceptive implant
    - if additional contraceptive precautions have not been taken when starting the method at a time when they were required and if UPSI has taken place
    - if UPSI has taken place whilst taking enzyme-inducing drugs, or in the 28 days thereafter
- intrauterine system
    - UPSI in the five days prior to removal, perforation, complete or partial expulsion
    - if additional contraceptive precautions not taken when starting the method at a time when it was required and UPSI has taken place

- history
    - time elapsed since unprotected intercourse
    - contraception used at the time of intercourse, if any
    - menstrual history
        - date of last menstrual period
        - whether last period was normal
        - usual cycle length
        - whether ovulation is likely to have taken place this cycle (around day 14 of 28 day cycle)
        - whether implantation of a fertilised ovum could have occurred this cycle (implantation occurs no earlier than 5 days after ovulation)
    - whether there has been any other unprotected intercourse this cycle and whether the woman might already be pregnant
    - any previous use of emergency contraceptives
    - whether she is breastfeeding

- obstetric and gynaecological history
    - including history of PID, current vaginal discharge, history of ectopic pregnancy
- current requirement for contraception
- medications used
    - e.g. enzyme inducing medications such as phenytoin and OTC such as St John's wort
- general health
    - including contraindications such as liver disease, porphyria
- sexual history
    - including risk of infection

- progestogen only emergency contraception – levonorgestrel
    - mode of action
        - thought to inhibit ovulation if used early in the cycle
        - unclear method of action later in the cycle
    - prescribing
        - LNG 1.5mg, taken as soon as possible after UPSI
        - available to buy over the counter for over 16s
        - several available brands, some are prescription only
    - timing
        - licenced for within 72 hours of UPSI
        - use beyond this is unlicensed but can be considered between 72-120 hours if other forms cannot be used and may have some efficacy
        - can be used more than once in a cycle if needed
        - repeated use will not induce abortion if the woman is already pregnant
        - can be safely used during lactation
    - efficacy
        - rates are difficult to determine but efficacy declines sharply by day 5
        - higher risk of failure rate in women taking enzyme-inducing drugs
            - if they do use LNG they should take a double dose (unlicensed)
    - contra-indications
        - hypersensitivity to LNG
        - acute porphyria
        - severe liver disease
        - severe malabsorption syndromes (e.g. Crohn's) may impair efficacy
        - rare hereditary problems of lactose intolerance
        - caution if there is a history of ectopic pregnancy (not an absolute contraindication)
    - side effects
        - nausea
        - vomiting
        - menstrual irregularities
            - may be delayed or there may be spotting
        - dizziness
        - diarrhoea
        - breast tenderness
    - interactions
        - liver enzyme-inducing drugs, e.g.:

                - anticonvulsants such as carbamazepine, phenytoin, topiramate
                - antibiotics – rifabutin, rifampicin
                - St John's wort
                - antiretrovirals
            - ciclosporin (LNG may increase risk of toxicity by inhibiting metabolism)
            - selegiline (may cause toxicity)
            - tizanidine (may cause toxicity)
        - specific advice for women
            - if vomiting occurs within two hours of taking, a repeat dose is required
            - women should use additional precautions for 7 days after use, 3 days for progestogen only pill
            - normal pill taking regime should recommence within 12 hours
- selective progesterone receptor modulator – ulipristal acetate
    - mode of action
        - inhibition or delay of ovulation
        - in the mid-follicular phase the further development of follicles is suppressed
        - if given at the time of the LH surge, the rupture of follicles is inhibited
        - after the LH peak it is not effective in delaying follicular rupture
    - prescribing
        - one tablet (30mg) of UPA
    - timing
        - should be taken as soon as possible after UPSI but within 120 hours (five days)
    - efficacy
        - performance is similar to or more effective than POEC
        - may be reduced if on liver enzyme inducing medication or medication which raises normal gastric pH
    - contraindications
        - pregnancy must be excluded
        - breastfeeding should be avoided for one week as there is insufficient research on safety
        - severe liver disease
        - uncontrolled asthma
        - repeated use within the same menstrual cycle
    - side effects
        - vomiting
            - repeat dose and consider antiemetic if vomiting occurs within 2 hours of taking
            - nausea
            - dizziness
            - menstrual irregularities
            - abdominal pain
            - myalgia and back pain
            - pelvic pain
            - headache
            - mood disorders
    - interactions

        - medication which increases gastric pH (e.g. antacids, PPIs, H2-receptor antagonists)
            - may reduce efficacy of UPA
        - liver enzyme inducing drugs
        - contraceptives
            - may reduce efficacy of ongoing contraception by competing with progestogens for progesterone receptors
    - copper intrauterine contraceptive device
        - mode of action
            - thought to have an inhibitory effect on both fertilisation and implantation
            - fertilisation inhibition is through direct toxicity of copper on ovum and sperm and inhibiting sperm penetration of cervical mucus
            - inflammatory effects of implantation on the endometrium can prevent implantation of the fertilised egg
                - some women may have moral concerns about this; by law pregnancy begins at implantation not fertilisation
                - to ensure it is fitted before implantation begins, it should be fitted within the first 120 hours (5 days) following first UPSI in a cycle or within 5 days from earliest estimated date of ovulation
            - IUCDs containing at least 380mm^2 of copper are more effective
    - timing
        - can be used for up to 5 days after UPSI
        - can be beyond this as long as it is not beyond 5 days from ovulation
        - if there is a delay in fitting, POEC should be given in the interim
    - efficacy
        - pregnancy rates are significantly less than 1%
        - most effective form of emergency contraception and the only one to provide ongoing contraception if left in situ
    - contra-indications
        - puerperal sepsis and septic abortion
        - current PID
        - history of copper allergy or Wilson's disease
        - markedly distorted uterine cavity
        - gestational trophoblastic disease with persistent elevated beta hCG levels, or malignant disease
        - cervical or endometrial cancer
        - active infection with chlamydia or gonorrhoea
    - risk of PID
        - there is potential risk of PID with insertion after UPSI
        - women at higher risk of STI (age 25 or under, new sexual partner or one or more sexual partners in previous year) should be offered testing for chlamydia
            - prophylactic antibiotics should also be considered at time of insertion
    - other considerations

        - previous ectopic pregnancy is not a contraindication to emergency IUCD use
        - non-copper containing IUCDs are not recommended for emergency contraception as there is no evidence of their effectiveness
        - IUCD may be removed following the next menstrual period if not required as long-term contraception
        - may also be removed if hormonal contraception is started within the first five days of the next cycle
- general points
    - discuss and document information about the failure rate
    - provide written advice sheet
    - explain that the next period may be on time, early or late
    - explain that they require a pregnancy test if they have not had a normal period within seven days of their expected next period or if they have irregular bleeding
    - advise that they should see a doctor immediately if they develop lower abdominal pain (possibility of ectopic pregnancy)
    - advise on more definitive method of contraception for the future, with written information if possible
    - discussion on risk of STI with referral for full sexual health screen as appropriate
    - documentation of Fraser-ruling competence if indicated

SeP4 post-exposure prophylaxis

- background
    - PEP may be offered for exposure to:
        - hepatitis B
        - hepatitis C
        - HIV
- when to prescribe
    - risk of transmission of HIV:
        - 3 in 1000 percutaneous exposures
        - <1 in 1000 mucocutaneous exposures
    - factors to consider
        - preventing avoidable exposure is of prime importance
        - healthcare workers and students should receive education on the risks of occupational exposure and advice to follow if it occurs
        - the views of the exposed patient should be taken into account when considering PEP
        - PEP is up to 80% effective but requires rapid action
- risk assessment for occupational exposure
    - exposure
        - an exposure to potentially infected blood, tissue or bodily fluids through:
            - a percutaneous injury
            - contact with mucous membranes (including the eye)
            - contact with skin that is abraded, inflamed or otherwise non-intact
    - level of risk is assessed according to:
        - type of exposure
            - percutaneous >mucous membrane >skin exposure
                - skin exposure risk is very small and difficult to quantify

        - body fluid involved:
            - amniotic fluid
            - blood
                - carries the highest risk
            - cerebrospinal fluid
            - exudative or other tissue fluid from burns or skin lesions
            - human breast milk
            - pericardial fluid
            - peritoneal fluid
            - pleural fluid
            - saliva in association with dentistry (likely to be contaminated with blood)
            - semen
            - synovial fluid
            - unfixed human tissues and organs
            - vaginal secretions
            - any other body fluid if visibly blood stained
        - severity of exposure
        - disease status of the source patient
            - high risk if the disease is at a more advanced stage and if resistant strains of HIV involved
        - obtaining consent from the source patient can be ethically difficult and the clinician needs to comply with GMC guidance, and possibly the Human Tissue Act and Mental Capacity Act
        - retrospective reviews suggest the greatest risk to be from percutaneous exposure to HIV infected blood, especially if:
            - there was visible contamination of a device with blood
            - procedure involved placement in a vein or artery
            - injuries were deep
            - the course patient was suffering from terminal stages of HIV infection
            - injury was with a hollow-bore needle
- risk assessment for non-occupational exposure
    - types of contact, in descending order of risk:
        - blood transfusion
        - receptive anal intercourse
        - receptive vaginal intercourse
        - insertive vaginal intercourse
        - insertive anal intercourse
        - receptive oral sex
    - assessment of risk is more difficult, with information about the source less likely to be available
    - PEP is likely to be effective if:
        - the risk of HIV transmission is high
        - the exposure is unlikely to be repeated
        - PEP can be started promptly
        - good adherence is likely

    - British Association for Sexual Health and HIV recommendations
        - PEP should be given:
            - receptive anal sex
            - insertive anal sex
            - receptive vaginal sex
            - insertive vaginal sex
        - PEP should be considered:
            - fellatio with ejaculation
            - splash of semen into the eye
        - PEP not recommended:
            - fellatio without ejaculation
            - cunnilingus
- what to prescribe
    - antiretroviral agents from three classes are currently licensed for treatment of HIV:
        - nucleoside/nucleotide analogue reverse transcriptase inhibitors (NRTIs)
        - non-nucleoside reverse transcriptase inhibitors (NNRTIs)
        - protease inhibitors (PIs)
    - no antiretroviral agent is licensed for PEP so they are used off label
    - DH recommended starting regime:
        - one Truvada tablet once a day
            - 245mg tenofovir and 200mg emtricitabine (NRTIs)
        - two Kaletra film-coated tablets twice a day
            - 200mg lopinavir and 50mg ritonavir (PIs)
    - Expert Advisory Group for AIDS preferred first line PEP regimen:
        - one Truvada tablet once a day for 28 days
        - one raltegravir tablet twice a day for 28 days (better tolerated than Kaletra so improves adherence)
    - regimes suitable for both occupational and non-occupational exposures
    - completion of 4 weeks of treatment is the goal
        - 50% have significant adverse effects and 30% stop taking PEP because of this
    - choice of drug is more limited in pregnancy
- monitoring and follow up
    - HIV testing (antibody with enzyme immunoassay) at twelve weeks after the exposure event or, if PEP was taken, at least 12 weeks after PEP was stopped
    - a longer period of monitoring is no longer recommended (not supported by evidence) – unless:
        - the individual is immunocompromised
        - the illness is compatible with an acute retroviral syndrome (regardless of the interval since exposure)
        - the source patient is dually infected
            - delayed seroconversion for HIV (at 7 months) has been reported in the case of HIV and HCV coinfection
    - counselling and safe sex education as indicated
    - prompt referral for treatment if patients become HIV positive
    - monitoring for toxicity of PEP with baseline FBC, U&E, LFTs
    - treatment of symptoms such as nausea
- factors contributing to failure of post-exposure prophylaxis

- delay in initiation of PEP
- large inoculum
- resistant strains of HIV
- short duration of PEP
- host factors
- non-adherence to regime

SeC2 sexual assault

- background
  - rape and sexual assault are common
  - lifetime risk of sexual violence for a woman is one in three
  - lifetime risk of rape or attempted rape for a woman is one in five
  - the majority of victims know their assailant
  - domestic violence is strongly associated with sexual assault and rape
- legal definitions
  - rape
    - penetration of the vagina, anus or mouth by a penis, without consent
    - both men and women can be raped
  - assault by penetration
    - the penetration of the vagina or anus with an object or body part, without consent
  - sexual assault
    - rape or assault by penetration including attempts are 'serious'
    - indecent exposure or unwanted touching are 'less serious'
- people at greater risk of sexual assault and rape
  - survivors of childhood or adolescent sexual or physical abuse
  - adolescents
  - young women
    - sexual assault four times more likely in the 16-24 age group
  - people with disabilities
  - people with substance abuse problems
  - homeless people
  - sex workers
  - prisoners and women in detention centres
  - people in institutions, including the military
  - people in areas of military conflict
- immediate care
  - have a level of suspicion and consider asking young people 'could you have said no?'
  - ideally, patients should be referred to a SARC (Sexual Assault Referral Centre)
    - this can be done whether or not they wish to report the assault to the police
    - collection of evidence can be done in an environment that avoids DNA contamination
    - victims can choose to be dealt with anonymously
    - evidence collected can subsequently be used in a legal case
  - forensic evidence should only be collected by trained personnel
    - the most fragile evidence, lost within 24 hours, is in the mouth

        - it can be collected in an early evidence kit and should be considered even if the victim does not want to involve the police, as they may change their mind later
        - a mouth rinse and urine sample collection for drugs and alcohol which may have facilitated the assault are available from police and can be stored without any immediate decision needing to be made about referral to SARC or reporting to the police
    - physical injuries should be treated as needed
- short-term effects
    - risk of pregnancy and need for emergency contraception should be assessed
    - risk of STI
    - emotional effects
        - most people experience profound emotional reactions in the weeks following a sexual assault
        - around 50% recover at 12 weeks but for many the symptoms persist for years
            - depression
                - significant risk of attempted and completed suicide
            - post traumatic stress disorder
                - more likely following rape than any other crime
                - early intervention may be indicated although evidence is lacking
- sexually transmitted infection risk and PEPSE
    - levels of risk are difficult to quantify but if there is any doubt, prophylaxis for STIs should be provided
    - PEPSE
        - antiretroviral triple therapy taken within 72 hours of exposure to HIV and continued for four weeks
        - side effects include nausea, diarrhoea, headache, tiredness
        - five day starter packs should have antiemetics and antidiarrhoeals included
        - regular blood tests are required due to the potential toxicity
        - risk of transmission = risk that source is HIV positive x risk of exposure
            - data to calculate this is available in the BASHH guidelines
            - treatment is advised if risk > 1 in 1000
                - risk of HIV transmission by receptive vaginal intercourse where there is no assault but the man is known to be HIV positive is 1 in 1000 per exposure
                    - with a heterosexual man of unknown risk born in the UK it is 1 in 250,000
- long term effects
    - disclosing previous abuse may take decades, with patients more likely to reveal it if asked directly
    - previous sexual abuse may be associated with:
        - pelvic pain
        - other chronic pain syndromes
        - fibromyalgia
        - chronic headaches

- PTSD is more likely if:
    - there was a perceived threat to life
    - violent force was used
    - reaction to disclosure was negative
    - the victim was from an ethnic minority
    - there had been previous abuse
        - greatest risk is for assaulted adults who were also assaulted in childhood or adolescence
    - previous mental health problems

SeC3 sexually transmitted infections

- risk factors
    - multiple partners
    - young age
    - failure to use barrier contraceptives
    - non-regular sexual relationships
    - men having sex with men
    - intravenous drug use
    - sub-Saharan African origin
    - social deprivation
    - prostitution
    - poor access to advice on and treatment of STIs
- history
    - core sexual history components (BASHH guidelines)
        - reasons for attendance; assess symptoms to guide examination and investigations required
        - exposure history to guide examination and investigations:
            - last sexual intercourse and number of partners in preceding three months
            - partner(s): gender, known or suspected infections, sites of exposure, condom use
            - previous STIs
        - for women:
            - assessment of contraceptive use and risk of pregnancy
            - last menstrual period
            - contraceptive use
        - assessment of other sexual health issues, including psychosexual problems
        - assessment of HIV, hepatitis B and hepatitis C risk for treatment and prevention purposes
        - assessment of risk behaviour for health promotion purposes
            - include alcohol and recreational drug use
        - past medical and surgical history, allergies and medication; cervical cytology history in women
        - establish mode of giving results
        - establish competency/child protection concerns if under 16
        - be aware of vulnerable adults, intimate-partner violence and gender-based violence

- symptom review
  - in women
    - change in vaginal discharge
    - vulval skin problems
    - lower abdominal pain
    - dysuria
    - changes in menstrual cycle or irregular bleeding
  - in men
    - urethral discharge
    - dysuria
    - genital skin problems
    - testicular swelling or discomfort
    - peri-anal/anal symptoms
- partners
  - establish if the patient is sexually active
    - all partners within the preceding three months
    - if no partners during this time, the last time they were sexually active should be recorded
    - if symptomatic, the sexual history should cover all partners during the incubation period of STIs that may cause current symptoms
    - establish relationship with partner (e.g. live-in, casual, paid) to assess risk and facilitate partner notification
    - where no unprotected oral, vaginal or anal sex is reported during this period, ask when the last time it was that this took place
  - check the relationship of symptoms to last sexual intercourse or to intercourse with a particular partner
  - condom use – whether always, sometimes or never
  - type of sex – e.g. oral, vaginal, anal
  - symptoms or diagnosis of partner(s)
  - sex with same or opposite sex partners (ever)
  - sex work – ask if they have ever been paid for sex
  - partners from overseas in the preceding year
- menstrual history and contraception
  - if contraception has been used, and if so, what method
  - check correct usage
  - last menstrual period and last sexual intercourse in relation to cycle – establish possibility of pregnancy
  - menstrual abnormalities – intermenstrual or post-coital bleeding
- previous STIs
  - previous diagnoses (and dates)
  - treatment
  - compliance
  - treatment of partner – consider risk of reinfection
- psychosexual history (where a sexual 'problem' or dysfunction identified)
  - establish how the patient sees the problem and what they consider the cause to be

        - duration of the problem and whether it is related to the time, place or partner
        - loss of sex drive or dislike of sexual contact
        - sources of stress, anxiety, guilt or anger
        - physical problems such as pain
        - illnesses that might affect sexual performance
            - e.g. testosterone or thyroid deficiency, pelvic or spinal trauma/surgery, arthritis
        - possibility of sexual abuse
    - other
        - past medical and surgical history
        - current medication, including over the counter and recreational drugs
        - allergies
        - HPV vaccination history in women born after 1995
        - history of cervical cytology in women
        - smoking and alcohol use
        - intravenous drug use with needle sharing (ever) and last use
- examination
    - females
        - external genital examination
            - inspect the perineum and anus
            - look around the genitals and between the vulval folds for:
                - lumps
                - swelling
                - lymphadenopathy
                - abnormal discharge
                - sores
                - ulcers
                - tears
                - scars
        - speculum examination for:
            - vaginal discharge and redness of the vaginal walls (vaginitis)
            - ulcers, sores or blisters
            - cervical abnormalities
                - tumours
                - contact bleeding
                - discharge
        - bimanual examination
            - lower abdominal tenderness (when pressing with outside hand)
            - cervical motion tenderness (often evident from facial expression) when the cervix is moved from side to side with the fingers of the gloved hand in the vagina
            - uterine or adnexal tenderness when pressing the outside and inside hands together
            - any abnormal swelling
                - e.g. pregnancy, uterovaginal prolapse, ovarian cysts, tumours

- males
  - ask the patient to stand and lower underpants to knees (or lie down as appropriate)
  - palpate the inguinal region for enlarged nodes or buboes
  - palpate the scrotum, feeling for the testis, epididymis and spermatic cord on each side
  - examine the penis, noting any rashes or sores
  - ask the patient to pull back the foreskin if present and look at the glans penis and urethral meatus
  - if there is no obvious discharge, ask the patient to milk the urethra
  - ask the patient to turn his back to you and bend over spreading his buttocks slightly; can also be done with the patient lying on his side with the top leg flexed up to his chest
    - examine the anus for ulcers, warts, rashes or discharge

- syphilis
  - background
    - caused by spirochete Treponema pallidum
    - nicknamed 'the great imitator' as causes signs and symptoms similar to other diseases
    - has symptomatic and asymptomatic stages
    - sexually transmitted by direct contact with syphilis ulcers or by infected blood through microtraumas during sexual intercourse
    - can be transmitted through vaginal, anal or oral sex
    - can be transmitted vertically during pregnancy
    - can be transmitted by needle sharing
  - signs and symptoms
    - average incubation period 21 days
    - primary syphilis
      - chancre(s) at the point of entry of the spirochete, resulting in a painless ulcer

https://dermnetnz.org/topics/syphilis/

      - typically a solitary small firm red painless papule on the genital area which quickly becomes a painless ulcer with well-defined margin and indurated base
      - multiple chancres in 30%
      - initial chancre may go unnoticed
      - non-tender local lymphadenopathy may occur

        - ulcer usually heals without treatment within a few weeks
- secondary syphilis
    - generalised infection within 3 months (average 6 weeks) of chancre
    - characterised by rash and systemic symptoms
        - fever
        - headache
        - malaise
        - myalgia
        - arthralgia
        - lymphadenopathy
    - patient is very infectious
    - symptoms will resolve untreated over a few weeks but can recur
    - other affected organs can include liver, kidneys, CNS, joints and eyes
    - cutaneous features:
        - non-itchy rash in 90%

https://dermnetnz.org/topics/syphilis/

            - subtle or rough, red or reddish-brown papules or plaques
            - typically occurs on trunk
            - frequently affects palms and soles
            - rarely presents as a cluster or papules around a central scaly plaque (corymbose syphilis)
            - patchy hair loss can occur
        - mucosal surfaces can become raw and red
        - greyish-white moist plaques occur in the groin, inner thighs, armpits, umbilicus or under the breasts (condyloma lata)

- latent syphilis
  - can be many years of latency between secondary and tertiary syphilis
  - patient is asymptomatic, infection only found on antibody tests
  - early latent syphilis (first two years)
    - patient is very infectious
    - can pass to sexual partners and from pregnant woman to foetus
  - late latent syphilis (after two years)
    - non-infectious to sexual partners
    - can pass from pregnant woman to foetus
- tertiary syphilis
  - late signs and symptoms can develop 20-40 years after initial infection in up to a third of non-treated cases
  - complications include endarteritis, gumma, cardiovascular and neurological disease
    - gumma
      - solitary granulomatous lesion with central necrosis
      - typically on skin or bone but can be found anywhere
      - skin gummas can be painless, gummas in long bones can cause a deep, boring pain that is worse at night
    - cardiovascular disease
      - rare complication
      - can causes aortic aneurysm and aortic regurgitation
    - neurosyphilis
      - can present as:
        - meningovascular disease
          - endarteritis leading to ischaemic stroke
        - general paresis
          - neuronal loss in the cortex leading to progressive dementia
        - tabes dorsalis
          - wasting away of the spinal cord

- congenital syphilis
  - if untreated can lead to miscarriage, stillbirth or congenital infection
  - early congenital syphilis (first two years of life)
    - in first few weeks, effects resemble secondary syphilis with multi-organ infection
      - skin and mucosa
        - maculopapular rash
        - vesiculobullous lesions
        - mucous patches
        - condyloma lata
        - rhinitis due to inflamed mucous membrane in the nose
      - can have neurological, ocular and haematological involvement

    - late congenital syphilis (after two years)
        - presents similarly to tertiary gummatous syphilis with chronic persisting inflammation
        - often affects the eyes (interstitial keratitis), ears (deafness), bones, joints, CNS
        - characteristic signs include:
            - Hutchinson incisors (peg shaped, notched, wider apart)
            - mulberry molars (multiple rounded cusps on the permanent first molars)
            - typical facial appearance
                - frontal bossing
                - saddle nose
            - bowed sabre shins
            - swollen knees (Clutton joints)
- diagnosis
    - identification of T. pallidum spirochaetes by microscopy or PCR in samples from lesions or lymph nodes
    - skin biopsy
    - serological testing (most commonly used)
        - cannot distinguish between different types of treponemal infection or duration of infection
            - further tests can potentially identify active infection but clinical history is important for interpretation
            - false positives can occur (particularly in autoimmune disease, injecting drug use, pregnancy and older age) so repeat testing may be needed
    - CT/MRI and/or CSF analysis may also be required
    - patients should have a full sexual health screen including for HIV
- treatment
    - penicillin by injection
        - stat dose for infectious syphilis, longer doses for late latent and neurosyphilis
        - tetracyclines and cephalosporins are used in patients with penicillin allergy but are less reliable
        - pregnancy women with penicillin allergy should be desensitised and treated with penicillin
        - treatment failures can occur at any stage, follow up and repeated testing are required
        - the patient should not have sexual activity until all sores or lesions are completely healed and treatment completed
        - syphilis can be acquired repeatedly despite antibodies
- gonorrhoea
    - background
        - Gram-negative diplococcus infecting mucous membranes of the urethra, endocervix, rectum, pharynx and conjunctiva
        - transmission occurs by direct inoculation of infected secretions from one mucous membrane to another, usually sexually and occasionally perinatally

        - incubation period is usually 2-5 days but may be up to 10 days
        - resistance to antibiotics varies and can be spread rapidly by plasmid transfer of antibiotic resistant genes
        - mortality is rare, but morbidity, primarily pelvic inflammatory disease, is common
    - risk factors
        - young age
        - history of previous STI
        - co-existent STIs
        - new or multiple sexual partners
        - recent sexual activity abroad
        - certain sexual activities – e.g. anal intercourse, frequent insertive oral sex
        - inconsistent condom use
        - history of drug use or commercial sex work
    - presentation
        - symptomatic in most men and asymptomatic in half of women
        - men
            - urethral infection
                - discharge (>90%)
                - dysuria (>50%)
                - asymptomatic (<10%)
            - rectal infection
                - usually asymptomatic
                - anal discharge (12%)
                - perianal/anal pain, pruritus, bleeding (7%)
            - pharyngeal infection
                - usually asymptomatic (>90%)
        - women
            - endocervical infection
                - frequently asymptomatic (up to 50%)
                - increased or altered vaginal discharge (most common symptom, up to 50%)
                - lower abdominal pain (up to 25%)
                - intermenstrual bleeding/menorrhagia (rare)
            - urethral infection
                - dysuria (10-15%) without frequency
            - rectal infection
                - spread by infected genital secretions or anal intercourse
                - usually asymptomatic
            - pharyngeal infection
                - usually asymptomatic (>90%)
    - signs
        - men
            - mucopurulent or purulent urethral discharge
                - appears +/- dysuria 2-5 days after exposure
            - epididymal tenderness/swelling or balanitis (rare)
        - women

- mucopurulent endocervical discharge (not a sensitive predictor of infection)
- easily induced contact bleeding of the endocervix
- pelvic/abdominal tenderness (uncommon, 5%)
- normal examination (very common

- children
  - acute conjunctivitis with purulent discharge
    - usually bilateral
    - <48 hours after birth
    - often with chemosis and lid oedema
  - vaginal discharge and vulval erythema

- haematogenous dissemination
  - causes skin lesions, arthralgia, arthritis, tenosynovitis (disseminated gonococcal infection
- management
  - GUM referral
  - safe sex advice and advice to avoid unprotected intercourse until 7 days after treatment finished
  - medication
    - ceftriaxone 1g IM single dose when sensitivities unknown
    - ciprofloxacin 500mg PO as single dose where sensitivities known
- complications
  - men
    - gonococcal urethritis
      - can cause urethral scarring and stricture resulting in bladder outflow obstruction
    - local spread
      - causes acute epididymitis, prostatitis, seminal vesiculitis, penile lymphangitis, peri-urethral abscess, infection of Tyson's and Cowper's glands
  - women
    - Bartholin's abscess
    - PID
      - thought to occur in 10-20%
      - causes infertility, chronic pelvic pain, ectopic pregnancy
    - peri-hepatitis (Fitz-Hugh Curtis syndrome)
    - premature labour and miscarriage
    - corneal scarring and severe sight impairment from neonatal ophthalmic infection
  - general
    - haematogenous dissemination (uncommon)
      - skin lesions (papules, bullae, petechiae, necrotic skin lesions
      - arthralgia, arthritis and tenosynovitis of the ankles, wrists, hands and feet (reactive arthritis)
      - meningitis, endocarditis or myocarditis with risk of death or permanent sequelae
    - increased risk of acquiring and transmitting HIV

- chlamydia
    - background
        - obligate intracellular Gram-negative bacteria that infect columnar and transitional epithelium
        - responsible for:
            - ocular infection (trachoma)
            - genitourinary infections
            - proctitis
            - sexually acquired reactive arthritis
            - lymphogranuloma venereum (rare sexually transmitted tropical infection causing genital ulcers and inguinal lymphadenopathy
        - most commonly diagnosed STI in the UK
        - asymptomatic in 50% of men and 70% of women
        - highest prevalence in sexually active people under 25
    - risk factors
        - age <25
        - sexual partner positive for chlamydia
        - two or more sexual partners in the preceding year
        - recent change in sexual partner
        - lack of consistent use of condoms
        - non-barrier contraception
        - infection with another STI
        - poor socio-economic status
        - genetic predisposition
    - symptoms
        - usually asymptomatic and detected at screening
        - female
            - vaginal discharge
            - dysuria
                - chlamydia should always be considered as a cause of sterile pyuria
            - vague lower abdominal pain
            - fever
            - intermenstrual or postcoital bleeding
            - deep dyspareunia
        - men
            - urethritis with dysuria and urethral discharge
            - epididymo-orchitis presenting as unilateral testicular pain +/- swelling
            - fever
        - both sexes
            - triad of urethritis, arthritis, conjunctivitis
            - upper abdominal pain due to perihepatitis
            - proctitis with mucopurulent discharge – may be due to rectal chlamydia during anal intercourse
            - pharyngeal infection
    - signs

        - female
            - friable, inflamed cervix
                - sometimes with a follicular or ‘cobblestone’ appearance
                - contact bleeding
            - mucopurulent endocervical discharge
            - abdominal tenderness
            - pelvic adnexal tenderness on bimanual palpation
            - cervical excitation
        - male
            - epididymal tenderness
            - mucoid or mucopurulent discharge
            - perineal fullness due to prostatitis
    - management
        - referral to GUM clinic for testing
            - recommendations for testing include:
                - symptoms that could indicate infection
                - sexual partners of people with proven or suspected chlamydia
                - all sexually active people under 25 annually, or more frequently if they have changed partner
                - people under 25 treated for chlamydia in the last 3 months
                - people with concerns about sexual exposure
                - people who have had two or more sexual partners in the past year
                - all women presenting for termination of pregnancy
                - all people presenting at GUM clinic
                - mothers of infants with chlamydial infection
                - women about to be fitted with an intrauterine contraceptive device who are at risk of STI
        - antibiotics
            - doxycycline 100mg BD (contraindicated in pregnancy) OR
            - single dose 1g azithromycin
        - safeguarding for children under 16 without evidence of vertical transmission
    - prognosis
        - spontaneous resolution of untreated chlamydia in up to 95% in four years
        - rates of progression to PID and infertility is unknown
        - antibiotic treatment is effective in 95% if the course is completed
        - two thirds of sexual partners will also test positive

SURGICAL EMERGENCIES

SuP1 abdominal pain

- background
    - acute abdomen is abdominal pain in the presence of tenderness and rigidity and is a surgical emergency

- it is important to remember that the abdominal wall can be a source of pain, and that the pain can come from above the diaphragm

- causes
  - generalised abdominal pain with tenderness and rigidity
    - perforated peptic ulcer
    - perforation of other intra-abdominal organs
    - ruptured AAA
    - aortic dissection
    - pancreatitis
    - ectopic pregnancy
    - ischaemic gut
  - central/peri-umbilical pain without abdominal signs
    - acute small bowel ischaemia
    - acute appendicitis
    - acute small bowel obstruction
    - testicular torsion
    - medical causes of abdominal pain
  - epigastric pain
    - gastrointestinal
      - gastritis
      - peptic ulcer
      - reflux oesophagitis
      - pancreatitis
      - cancer (gastric, pancreatic)
      - Boerhaave syndrome
      - dyspepsia
      - irritable bowel syndrome
    - pain from nearby areas
      - cardiac
        - myocardial infection
        - pleuritis
      - pulmonary
        - pneumonia
        - pleurisy
  - right hypochondrium/upper quadrant pain
    - gall bladder
      - biliary colic
      - cholecystitis
      - cholangitis
    - liver
      - hepatitis
      - hepatomegaly (e.g. liver congestion in right heart failure)
      - haemorrhage into hepatic tumour
      - trauma
      - hepatic or subdiaphragmatic abscess
      - Fitz-Hugh-Curtis syndrome (peri-hepatitis due to PID)
      - pre-eclampsia and HELLP

        - other gastrointestinal
            - appendicitis with high appendix (e.g. pregnancy)
            - perforated or penetrating duodenal ulcer
            - colon cancer
        - pain from nearby areas
            - lung
                - right lower lobe pneumonia
                - pleurisy
                - other lung disease
            - subphrenic abscess
            - acute pyelonephritis
    - left hypochondrium/upper quadrant pain
        - pancreatitis
        - subphrenic abscess
        - diverticulitis
        - ruptured spleen
        - acute pyelonephritis
        - leaking aneurysm of the splenic artery
        - acute gastric distension
    - right iliac fossa pain
        - gastrointestinal
            - appendicitis
            - Crohn's disease
            - inflamed Meckel's diverticulum
            - cholecystitis with low gallbladder
            - mesenteric adenitis
            - epiploic appendagitis
            - colon cancer
            - constipation
            - irritable bowel syndrome
        - reproductive (female)
            - ectopic pregnancy
            - acute ovarian event
                - cyst rupture
                - haemorrhage
                - torsion
            - Mittelschmerz
            - pelvic inflammatory disease
            - endometriosis
        - reproductive (male)
            - seminal vesiculitis
            - undescended testicle pathology
        - urinary
            - renal colic
            - UTI
        - pain from nearby areas
            - hip pathology

                - psoas abscess
                - rectus sheath haematoma
                - right lower lobe pneumonia
    - left iliac fossa pain
        - gastrointestinal
            - diverticulitis
            - colitis
            - colon cancer
            - constipation
            - irritable bowel syndrome
        - reproductive (female)
            - ectopic pregnancy
            - acute ovarian event
                - cyst rupture
                - haemorrhage
                - torsion
            - Mittelschmerz
            - pelvic inflammatory disease
            - endometriosis
        - reproductive (male)
            - seminal vesiculitis
            - undescended testicle pathology
        - urinary
            - renal colic
            - UTI
        - pain from nearby areas
            - hip pathology
            - psoas abscess
            - rectus sheath haematoma
            - left lower lobe pneumonia
    - suprapubic pain
        - urinary retention
        - cystitis
        - uterine in origin
            - PID
            - fibroid
            - menstruation
        - origin from RIF or LIF causes
    - loin pain
        - renal tract
            - infection (e.g. pyelonephritis)
            - obstruction (e.g. renal colic)
            - renal carcinoma
            - renal vein thrombosis
            - polycystic kidney disease
            - adrenal haemorrhage
        - other

                - retroperitoneal haemorrhage
                - retroperitoneal infection
                - vertebral pathology
        - groin pain
            - renal calculi
            - scrotal pain
                - testicular torsion
                - epididymo-orchitis
                - trauma
            - inguinal hernia
            - hip pathology
            - pelvic fracture

SuP2 abdominal swelling/mass

- background
    - distention: diffuse swelling or enlargement of the abdomen
    - distension can also apply to the sensation of elevated abdominal pressure and volume
- causes
    - fat
        - obesity
    - flatus
        - intestinal obstruction
    - foetus
        - pregnancy
    - fluid
        - ascites
        - distended bladder
        - fluid in obstructed bowel
    - faeces
        - constipation
        - faecal impaction
    - filthy great tumour
        - ovarian cyst
        - lymphoma
    - flippin' big organs
        - organomegaly
    - foreign body

SuP3 constipation

- background
    - chronic constipation (>6 months) is common with a prevalence of 14% worldwide
    - prevalence is higher in women and people of low socio-economic status
- definition
    - constipation is a symptom, not a diagnosis
    - patients must be asked what exactly they mean by it

    - defined as defaecation that is unsatisfactory because of infrequent stools (<3 times/week), difficult stool passage (with straining or discomfort) or seemingly incomplete defaecation
    - stools are often dry and hard and may be abnormally large or abnormally small
- history
    - frequency, nature and consistency of stool
    - blood or mucus in/on the stool
    - whether there is diarrhoea alternating with constipation
    - any recent change in bowel habit
    - diet
    - drugs
- causes
    - common
        - low-fibre diet
        - inadequate fluid intake or dehydration
        - immobility or lack of exercise
        - irritable bowel syndrome
        - elderly age
        - postoperative pain
        - hospital environment (lack of privacy, having to use a bedpan)
    - anorectal disease
        - anal fissure
        - anal stricture
        - rectal prolapse
    - intestinal obstruction
        - strictures (e.g. Crohn's disease)
        - colorectal carcinoma
        - pelvic mass (e.g. foetus, fibroids)
        - diverticulosis
        - congenital abnormalities
        - pseudo-obstruction
    - metabolic/endocrine
        - hypothyroidism
        - hypercalcaemia
        - hypokalaemia
        - porphyria
        - lead poisoning
    - drugs
        - opioid analgesics
        - anticholinergics
        - iron
    - neuromuscular
        - spinal or pelvic nerve injury
        - American trypanosomiasis (Chagas disease)
        - Hirschsprung's disease
        - systemic sclerosis
        - diabetic neuropathy
    - other

        - chronic laxative abuse (rare – diarrhoea is more common)
        - idiopathic slow transit
        - idiopathic megacolon/megarectum
- investigations
    - most constipation does not require investigation, especially in young, mildly affected patients
    - indications for investigation include:
        - age >40
        - recent change in bowel habit
        - associated symptoms
            - weight loss
            - rectal bleeding
            - mucous discharge
            - tenesmus
    - possible investigations include:
        - bloods: FBC, U&E, TFTs, calcium
        - sigmoidoscopy and biopsy of normal and abnormal mucosa
        - barium enema if there is suspected colorectal malignancy
        - special investigations: e.g. transit studies, anorectal physiology
- management
    - treat the cause
    - mobilise the patient
    - increase fluid intake
    - increase intake or high-fibre foods
    - consider drugs if the above measures fail
        - bulk producers
            - increase faecal mass, which stimulates peristalsis
            - must be taken with plenty of fluid
            - contra-indications:
                - difficulty in swallowing
                - intestinal obstruction
                - colonic atony
                - faecal impaction
            - examples:
                - bran powder
                - ispaghula husk
                - methylcellulose
                - sterculia
        - stool softeners
            - side-effects can include:
                - anal seepage
                - lipoid pneumonia
                - malabsorption of fat soluble vitamins
            - examples:
                - arachis oil enemas
                    - lubricate and soften impacted faeces
                - liquid paraffin

        - should not be used for a prolonged period
    - stimulants
        - increase intestinal motility
        - should not be used in intestinal obstruction
        - prolonged use should be avoided – may cause colonic atony and hypokalaemia (but no good long-term follow-up studies)
        - examples:
            - pure stimulant laxatives are bisocodyl tablets (5-10mg ON) or suppositories (10mg OM) and senna 2-4 tablets at night
            - docusate sodium and dantron have stimulant and softening actions
                - dantron is associated with colonic and liver tumours in animals so only used in the elderly or terminally ill
            - glyceryl suppositories act as a rectal stimulant
            - sodium picosulfate is useful for rapid bowel evacuation prior to a procedure
    - osmotic agents
        - retain fluid in the bowel
        - examples:
            - lactulose
                - semisynthetic disaccharide which produces an osmotic diarrhoea of low faecal pH that discourages growth of ammonia producing organisms
                - useful in constipation (15ml BD) and hepatic encephalopathy (30-50ml BD)
            - magnesium salts
                - e.g. magnesium hydroxide and magnesium sulfate
                - useful when rapid bowel evacuation is required
            - macrogols
                - can be used in the long-term management of chronic constipation
    - enemas and suppositories
        - useful additional treatment
        - examples:
            - sodium phosphate enemas and glycerin suppositories
            - avoid sodium salts – can cause sodium and water retention
            - phosphate enemas are useful for rapid bowel evacuation prior to procedures
            - excessive use of soapy tap water enemas may lead to water intoxication

- cost
    - cheap
        - senna, bran, co-danthrusate, bisacodyl
    - moderate
        - magnesium hydroxide, methylcellulose, ispaghula granules, sterculia
    - expensive
        - lactulose

- prucalopride
    - selective serotonin receptor agonist with prokinetic properties
    - recommended for prescription by clinicians experienced in treating chronic constipation for women for whom treatment with laxatives from two different classes at the highest dose possible for at least six months has failed
    - only recommended for women as lack of data on use in men
- obstructed defecation syndrome
    - characterised by an urge to defecate but an inability to expel the faecal bolus
    - symptoms include:
        - unsuccessful attempts at faecal evacuation
        - excessive straining
        - pain
        - bleeding after defecation
        - sense of incomplete faecal evacuation
    - more common in women, particularly multiparous women
    - often associated with structural defects in the rectum, such as rectocele, internal rectal prolapse and perineal descent
    - conservative treatments include:
        - diet
        - biofeedback
        - laxatives
        - pelvic floor retraining
    - surgery
        - may be considered for patients not responding to conservative management or if structural abnormalities are present
        - options include stapled transanal prolapsectomy, perineal levatorplasty (STAPL), stapled transanal rectal resection (STARR) and laparoscopic ventral mesh sacrocolporectopexy
- constipation in children
    - background
        - affects 5-30% of children
        - often accompanied by parental anxiety
        - typically characterised by infrequent bowel evacuations, large stools and difficult or painful evacuation
        - referral to secondary care for chronic symptoms is common
        - 90-95% is functional
        - similar prevalence in boys and girls
        - commonly seen in:
            - infants at weaning
            - toddlers acquiring toilet skills
            - school age
        - may also be associated with problems with toilet training, psychological problems, major life events, neurodevelopmental disorders, autism
    - diagnostic criteria (Paris Consensus on Childhood Constipation Terminology – PACCT – Group)
        - chronic constipation
            - fewer than 3 bowel movements per week

- more than one episode of faecal incontinence per week
- either palpable stools in the abdomen or large stools palpable rectally
- passing stools so large they block the toilet
- retentive posturing and withholding behaviours
- painful defecation

- faecal incontinence (passage of stool in inappropriate places)
  - organic faecal incontinence
    - faecal incontinence resulting from organic disease
  - functional faecal incontinence
    - faecal incontinence without organic disease
      - constipation associated faecal incontinence
      - non-retentive faecal incontinence
        - passage of stools in inappropriate places in children over 4 with no associated constipation
- faecal impaction
  - large faecal mass (abdominal or rectal) unlikely to be passed on demand
- pelvic floor dyssynergia
  - inability to relax the pelvic floor when attempting to defecate

- presentation
  - history
    - past medical, school and social history
    - family history
    - frequency of defecation
    - consistency of stools – may include use of Bristol Stool Chart
    - episodes of faecal incontinence
    - pain on defecation
    - whether stools block the toilet
    - any associated behaviour
      - pain on defecation is likely to lead to withholding
  - examination
    - palpation of the abdomen for faecal mass
    - inspection for anal stenosis or ectopia
    - sacral abnormalities
    - rectal examination is not routinely necessary
- causes
  - organic causes (mostly occur within the first few weeks of life)
    - anorectal malformations
      - requires careful inspection of the perineum in any baby with constipation
    - anal fissure
      - painful defecation
      - passage of blood and sentinel pile on anterior anus
    - rectal prolapse

        - can be caused by chronic straining and constipation, disorders of sacral nerve innervation, chronic diarrhoea
    - Hirschsprung's disease
        - usually presents early as delay in passing meconium or failure to thrive in the first month
        - can rarely present late
        - diagnosis is usually by rectal biopsy
    - neurenteric problems
        - test of choice is colonic motility
    - spinal cord problems
        - generally diagnosed by physical examination and MRI
    - pelvic floor dyssynergia
        - requires anorectal manometry
    - metabolic or systemic disorders
        - hypothyroidism
        - coeliac disease
        - hypocalcaemia
        - cystic fibrosis
    - toxic
        - lead levels
        - toxicology screen
    - cow's milk allergy
        - elimination diet
        - allergy testing
- functional causes
    - history and physical examination are generally sufficient for diagnosis
    - further investigations are occasionally required and always in non-retentive faecal incontinence

- diagnosis
    - children under 1
        - less than 3 complete stools per week (unless exclusively breastfed, where it can be normal)
        - large hard stool or 'rabbit droppings'
        - symptoms associated with defecation
            - distress on passing stool
            - bleeding with hard stool or straining
        - past history of constipation
        - previous or current anal fissure
    - older children may present with the above plus:
        - overflow soiling
            - child may be unaware
            - loose smelly stools, may be thick and sticky or dry and flaky
            - large stools, big enough to block the toilet
            - poor appetite that improves with passage of a large stool
            - abdominal pain which changes with passage of stool
            - retentive posturing

            - e.g. on tiptoes, straight legged with arched back
        - straining, painful bowel movements and/or anal pain
- red flags
    - symptoms that commence from birth or within the first few weeks
    - failure or delay (>first 48 hours at term) in passing meconium
    - ribbon stools
    - leg weakness or locomotor delay
    - abdominal distension with vomiting
    - abnormal examination findings including:
        - abnormal appearance of anus
        - gross abdominal distension
        - abnormal gluteal muscles, scoliosis, sacral agenesis etc.
        - limb deformity including talipes
        - abnormal reflexes
    - management:
        - do not treat constipation
        - urgent referral to appropriate specialist
- amber flags
    - constipation with faltering growth
    - possible maltreatment
    - management:
        - if there is evidence of faltering growth, treat for constipation and test for coeliac disease and hypothyroidism
        - if possible maltreatment, follow guidelines for safeguarding
- idiopathic constipation
    - exclude red and amber flags
    - history of meconium being passed within 48 hours of birth (full term baby)
    - constipation begins at least a few weeks after birth
    - precipitating factors may be present, such as weaning, poor fluid intake
    - abdomen is soft and not distended, normal appearance of anus
    - general health, growth and development are normal, with normal gait, tone and power in lower limbs
- management of functional/idiopathic constipation
    - aims
        - remove faecal impaction
        - restore bowel habit (with soft stools passed without pain)
        - self-toileting
        - passing of stools in appropriate places
    - address anxiety of parent and child
    - address attitudes of guilt and blame
    - address inappropriately coercive toilet training
    - social consequences (e.g. faecal incontinence in older children)
    - disimpaction
        - clearing of retained faeces from the rectum
        - initially, use of an osmotic laxative (e.g. Movicol paediatric)
            - may increase symptoms (e.g. soiling) at first
            - gradually increase dose if ineffective

        - if not tolerated, substitute stimulant laxative on its own or with lactulose (osmotic laxative) or faecal softener (docusate) if stools are hard
    - if ineffective after 2 weeks, add a stimulant laxative
        - e.g. sodium picosulfate or senna in children over 1 month, docusate (softener and weak stimulant) from 6 months or bisocodyl suppositories from 2 years
        - if unsuccessful, paediatrician referral
    - avoid rectal treatments in children (suppositories, enemas, manual evacuation), although they may be recommended by specialists
    - weekly review until successful
- maintenance therapy
    - dietary advice, including fluid and fibre intake
    - use of bowel charts and a diary
    - regular laxatives over months or even years, preferably osmotic (PEG 3350 or lactulose), titrated to maintain soft formed stool
    - avoid stopping and starting treatment causing intermittent impaction
    - avoid prolonged use of stimulant laxatives (risk of atonic colon, hypokalaemia) – use inly intermittently to avoid impaction
- modification of behaviour
    - specific techniques can be used in specialist clinics:
        - encourage regular, unhurried toileting
        - encourage use of reward systems for successful use of the toilet
        - encourage linkage of diary to reward system
- incontinence
    - explanation to the parent and child of the involuntary nature
    - encouragement of regular toileting
    - involvement of the school nurse if possible to help with toileting and teacher education

- indications for specialist review
    - organic cause suspected or any red flags
    - treatment unsuccessful (no response in four weeks for a child under 1 or three months in an older child)
    - management is complex
    - child abuse is suspected
- complications
    - faecal impaction
    - chronic constipation
    - megacolon (may predispose to, or result from, constipation)
    - rectal prolapse
    - anal fissure
    - faecal soiling
    - psychological effects
- prognosis
    - multiple relapses are common

        - resolution in 50% after one year and 65-70% after two years

SuP4 diarrhoea

- acute diarrhoea
    - background
        - no universally agreed definition
        - British Society of Gastroenterology definition is the abnormal passage of loose or liquid stools more than three times a day and/or a volume of stool greater than 200g/day
        - acute diarrhoea is normally defined as lasting less than 4 weeks
    - causes
        - infection
            - viral
                - e.g. norovirus, rotavirus, sapovirus
            - Clostridium difficile
                - common in older people who have taken antibiotics
            - infections that can present with bloody diarrhoea include:
                - cytomegalovirus
                - Campylobacter jejuni
                - Salmonella spp
                - Escherichia coli O157
                - Vibrio parahaemolyticus
                - Shigella spp
                - Yersinia spp
                - Aeromonas spp
                - C difficile
                - Entamoeba histolytica
                - schistosomiasis
                - Ebola virus
        - drugs associated with diarrhoea
            - allopurinol
            - antibiotics
            - digoxin
            - colchicine
            - cytotoxic drugs
            - magnesium-containing antacids
            - metformin
            - NSAIDs
            - PPIs
            - SSRIs
            - statins
            - theophylline
            - thyroxine
            - high dose vitamin C
        - constipation with overflow diarrhoea
        - other causes of acute diarrhoea:
            - anxiety

- food allergy
- acute appendicitis
- acute radiation enteritis
- intestinal ischaemia

- history
  - frequency and severity of symptoms
    - quantity and character of stools (watery, fatty, containing blood or mucus)
  - presence of red flag symptoms
    - blood in stool
    - recent hospital treatment or antibiotics
    - persistent vomiting
    - weight loss
    - painless, watery, high-volume diarrhoea (increased risk of dehydration)
    - nocturnal symptoms disturbing sleep (organic cause likely)
  - features suggesting infection
    - fever
    - recent travel abroad
    - contact with another person with diarrhoea
    - possible source of food poisoning
  - possible drug causes
  - stress or anxiety
  - clinical features suggesting appendicitis
  - recent radiotherapy
  - cardiovascular risk factors (intestinal ischaemia)
  - comorbidities that may increase risk of complications
    - heart disease
    - diabetes
    - CKD
    - immunodeficiency
- examination
  - signs and symptoms of dehydration
    - mild
      - lassitude
      - anorexia
      - nausea
      - light-headedness
      - postural hypotension
      - usually no signs
    - moderate dehydration
      - apathy
      - tiredness
      - dizziness
      - muscle cramps
      - dry tongue
      - sunken eyes

        - reduced skin elasticity
        - postural hypotension
        - tachycardia
        - oliguria
    - severe dehydration
        - profound apathy
        - weakness
        - confusion (leading to coma)
        - shock
        - tachycardia
        - marked peripheral vasoconstriction
        - systolic <90mmHg
        - oliguria or anuria
  - consider rectal examination, particularly in those aged 50 or older (faecal loading in the rectum, colorectal cancer)
- investigations
  - not always necessary for acute diarrhoea
  - stool specimen for culture and sensitivity if:
    - person is unwell, immunocompromised, recently received antibiotics or had recent hospital admission
    - blood or pus in stool
    - underlying cause uncertain or diarrhoea ongoing for more than a week
    - diarrhoea after foreign travel (also test for ova, cysts and parasites)
  - advice from local health protection unit if:
    - suspected public health hazard (e.g. food handlers, healthcare workers, elderly care home residents)
    - outbreaks of diarrhoea where isolating the organism might help pinpoint the source
    - contacts of people infected with organisms that may cause serious clinical sequelae (e.g. E. coli O157)
  - further investigation if suspected chronic cause
- management
  - supportive – prevention or reversal of fluid and electrolyte depletion
  - specific treatment for underlying cause if needed
  - symptomatic treatment
    - only where there is a clear underlying cause
    - antimotility drugs relieve symptoms of acute diarrhoea
    - antispasmodics may be helpful for cramps
- admission criteria may include:
  - vomiting and inability to retain oral fluids
  - features of severe dehydration or shock
  - older age
  - poor home circumstances and level of support
  - bloody diarrhoea
  - abdominal pain and tenderness (may suggest surgical cause)

        - increased risk of poor outcome (e.g. co-morbidities or drugs such as immunosuppressants or systemic steroids)
    - two week wait referral
        - ≥40 with unexplained weight loss and abdominal pain
        - ≥50 with unexplained rectal bleeding
        - ≥60 with iron-deficiency anaemia or changes in bowel habit or occult blood in faeces
        - rectal or abdominal mass
        - change in bowel habit
        - weight loss
        - iron-deficiency anaemia
    - complications
        - dehydration and electrolyte imbalance
        - reactive complications (e.g. arthritis)
        - spread of infection
        - irritable bowel syndrome
        - lactose intolerance
        - haemolytic uraemic syndrome
        - reduced drug absorption with potentially serious consequences (e.g. antiepileptic drugs, oral contraceptives)
- chronic diarrhoea
    - background
        - pragmatic definition is persistent alteration from the norm with stool consistency between types 5 and 7 on the Bristol Stool Chart and increased frequency for greater than four weeks in duration
        - persistent diarrhoea suggests a non-infectious aetiology and should be further investigated
        - malabsorption is suggested by steatorrhoea and the passage of bulky foul-smelling pale stools
        - colonic, inflammatory or secretory forms of diarrhoea typically present with liquid loose stools with blood or mucus discharge
    - causes
        - irritable bowel syndrome
        - bile acid malabsorption
        - colonic:
            - diverticular disease
            - colonic neoplasia
            - ulcerative colitis and Crohn's colitis
            - microscopic colitis
                - type of inflammatory bowel disease, normally presenting in older age groups with chronic watery diarrhoea
                - multiple colonic biopsies required for diagnosis
            - ischaemic colitis
            - constipation with overflow
        - small bowel
            - coeliac disease
            - Crohn's disease

- other small bowel enteropathies
    - e.g. Whipple's disease, tropical sprue, amyloid, intestinal lymphangiectasia
- bile acid malabsorption
- disaccharidase deficiency
- lactose intolerance
- small bowel bacterial overgrowth
- mesenteric ischaemia
- radiation enteritis
- lymphoma

- pancreatic
    - chronic pancreatitis
    - pancreatic carcinoma
    - cystic fibrosis
- endocrine
    - hyperthyroidism
    - diabetes and other causes of autonomic neuropathy
    - hypoparathyroidism
    - Addison's disease
    - hormone-secreting tumours (VIPoma, gastrinoma, carcinoid)
- chronic infection
    - e.g. amoebiasis, giardiasis, hookworm, Cryptosporidium spp, Entamoeba histolytica
- recent antibiotic therapy and C diff infection
- previous surgery
    - post-gastrectomy
    - extensive resections of the ileum and right colon
    - bacterial overgrowth, particularly in bypass operations
    - bile acid diarrhoea in shorter resections of the terminal ileum (usually responds to fasting and colestyramine)
    - cholecystectomy
- drugs
- food additives such as sorbitol or fructose
- alcohol
- immunodeficiency
- factitious diarrhoea (may be associated with an eating disorder)

- red flags
    - unintentional and unexplained weight loss
    - rectal bleeding
    - diarrhoea for more than 6 weeks in a patient over 60
    - family history of bowel or ovarian cancer
    - abdominal mass
    - rectal mass
    - anaemia
    - raised inflammatory markers
- investigations may include:
    - FBC (anaemia, raised platelet count)

        - LFTs
        - U&Es
        - tests for malabsorption (calcium, B12, folate, ferritin)
        - TFTs
        - ESR/CRP
        - antibody tests for coeliac disease
        - Ca125 if ovarian cancer suspected
        - HIV serology if immunodeficiency suspected
        - stool for culture, sensitivity, ova, cysts, parasites if infectious cause suspected or travel abroad
        - faecal calprotectin to distinguish between IBS and inflammatory bowel disease in people under 40
        - testing for blood in stool if symptoms suggestive of colorectal cancer and patient does not meet suspected cancer referral pathway
    - further specialist investigations may include:
        - small bowel
            - distal duodenal biopsies
            - barium follow-through
            - endoscopy
            - bacterial overgrowth: glucose hydrogen breath test, jejunal aspirate and culture
        - pancreatic
            - CT pancreas
            - faecal elastase or chymotrypsin
            - ERCP or MRCP
        - flexible sigmoidoscopy
        - colonoscopy
        - video capsule endoscopy
    - two week wait referral
        - as for acute diarrhoea
    - management
        - depends on underlying cause
        - may include antimotility drugs such as codeine, loperamide where a definite diagnosis has been made and there is no cause-associated contraindication
- acute diarrhoea in children
    - worrying features suggesting more than simple viral gastroenteritis include:
        - generally unwell child out of proportion to the level of dehydration
        - abdominal pain with tenderness and guarding (possible surgical problem)
        - shock, pallor, jaundice, poor urine output
        - bilious vomiting
        - blood in stool (possible intussusception or haemolytic uraemic syndrome)
    - causes
        - gastroenteritis
            - viral
                - mainly rotavirus, also norovirus, echoviruses and enteroviruses
            - bacterial

        - suggested by high fever and bloody diarrhoea
        - common pathogens include Shigella spp, Salmonella spp, Campylobacter spp
    - protozoal
        - e.g. giardia
- systemic infection
    - e.g. UTI, pneumonia, otitis media, meningitis, septicaemia
- antibiotic associated and rarely pseudomembranous colitis
- dietary
    - food allergy or intolerance
    - starvation stools
- surgical conditions
    - appendicitis
    - intussusception
    - partial bowel obstruction (e.g. volvulus, Hirschsprung's)
    - Meckel's diverticulum
    - short bowel syndrome
- malabsorption
    - cystic fibrosis
    - coeliac disease
- inflammation
    - ulcerative colitis
    - Crohn's disease
- miscellaneous
    - constipation with overflow
    - HUS
    - toddler's diarrhoea
    - hyperthyroidism

- history
    - patient may or may not be unwell with systemic symptoms
    - food history and whether anyone else in the family is affected
    - nonspecific non-focal abdominal pain and cramping are common
    - water exposure (swimming pools have been associated with outbreaks of shigellosis, giardiasis, cryptosporidiosis and amoebiasis)
    - travel history
    - animal exposure
- examination
    - signs of dehydration
    - shock – falling blood pressure, tachycardia, coma, anuria
    - evidence of failure to thrive/malnutrition
    - abdominal pain
        - does not increase with palpation
- investigations may include:
    - stool culture and ova/cysts/parasites
    - rotavirus antigen tests
    - WCC, U&Es
    - other investigations depending on the situation

                - e.g. endomysial antibodies (coeliac disease), intestinal biopsy (coeliac disease or inflammatory bowel disease), sweat test (cystic fibrosis)
        - management
            - supportive care with adequate oral rehydration
                - children under 6 months are at greatest risk of dehydration
            - breast feeding should be continued
            - loperamide reduces the duration but has not been proved to be safe and should not be advised
        - complications
            - rotavirus may cause dehydration and lactose intolerance
            - bacterial bowel infection rarely leads to infection at other sites; infection may also lead to bowel perforation, intussusception, appendicitis or liver abscess
            - rare complications include HUS and reactive arthritis
            - giardiasis may lead to chronic fat malabsorption
            - some patients may develop carrier states (e.g. rotavirus, salmonella)
- chronic diarrhoea (>2 weeks) in children
    - differential
        - well child with no weight loss
            - toddler's diarrhoea typically occurs in the second year of life and is associated with undigested food such as carrots and peas in the stool
                - child is well and growing normally
                - it is thought to relate to rapid intestinal transit time
                - resolves by the age of 4
            - breastfed babies often have liquid and apparently abnormal stools – does not require investigation if the baby is otherwise well and thriving
        - unwell child with weight loss
            - food intolerance (e.g. cow's milk protein intolerance)
            - inability to digest nutrients (e.g. pancreatic insufficiency, cystic fibrosis)
            - small intestinal disease (coeliac disease, food-sensitive enteropathy, inborn error of digestion or absorption, short bowel syndrome)
            - inflammatory bowel disease
            - intestinal parasites (e.g. Giardia)
            - chronic enteric infection (stagnant loop syndrome)
            - iatrogenic (therapeutic diets, drugs – e.g. excess laxatives)
            - miscellaneous (e.g. acrodermatitis enteropathica, immunodeficiency, Schwachman Diamond syndrome, hyperthyroidism)
    - investigations
        - as for acute diarrhoea
        - barium meal and follow through
            - look for malrotation or blind loop
        - endoscopy and biopsies

        - faecal calprotectin (recommended by NICE, may prevent need for endoscopy)

SuP5 GI bleeding

- upper gastrointestinal haemorrhage
    - background
        - incidence higher in elderly and low socio-economic status groups
        - upper GI bleed is defined as any bleeding which occurs from a source proximal to the ligament of Treitz (suspensory ligament of the duodenum which marks the duodenojejunal junction)
    - common causes in the UK
        - peptic ulcer disease (30%)
        - Mallory-Weiss tear (15%)
        - no diagnosis (15%)
        - erosion (10%)
        - oesophagitis (10%)
        - varices (10%)
        - other (5%)
        - vascular lesion (4%)
        - malignancy (1%)
    - history
        - haematemesis (present in 50%)
            - bleeds tend to be more severe than in those with only melaena
        - melaena (present in 70%)
        - dizziness/syncope
        - known or suspected liver disease
        - profuse recent vomiting (Mallory-Weiss tear)
        - previous peptic ulcer disease or gastritis
        - known or previous Helicobacter pylori infection
        - alcohol
        - medication (particularly NSAIDs, increase risk 4 fold)
    - examination
        - PR – evidence of melaena/haematochezia
        - hypotension
        - tachycardia
    - investigations may include:
        - VBG for quick assessment of Hb
        - U&Es (raised urea after significant bleed)
        - FBC
        - coag screen
        - LFTs
        - G&S/crossmatch depending on urgency
        - CXR
        - 12 lead ECG
    - management
        - haemodynamic instability
            - high concentration oxygen

- two large bore peripheral cannulae (IO or US guided if required)
- bloods
- transfusion as per local policy, with activation of major haemorrhage protocols early
- urinary catheter to measure output
- urgent referral to GI service
- involvement of critical care team

- blood transfusion
  - there are risks of overtransfusion
  - reduction in mortality with transfusion at 70 (90 with co-existing coronary heart disease)
  - platelet transfusion in actively bleeding patients with platelets <50 $x10^9$/L
  - FFP in actively bleeding patients with PT, INR or APTT >1.5 times the upper limit of normal
    - cryoprecipitate if fibrinogen remains <1.5g/L despite using FFP
  - prothrombin complex concentrate (PCC) for patients on warfarin who are actively bleeding
  - use of thromboelastography if available

- risk stratification
  - Glasgow-Blatchford score
    - useful in the ED
    - online calculators are helpful
    - NICE recommends that patients with a score of 0 can be considered for early discharge with outpatient endoscopy follow up
    - urea
      - 6.5-7.5 (2)
      - 8.0-9.9 (3)
      - 10.0-24.9 (4)
      - >25.0 (5)
    - Hb (men)
      - 120-129 (1)
      - 100-119 (3)
      - <100 (6)
    - Hb (women)
      - 100-119 (1)
      - <100 (6)
    - systolic BP
      - 100-109 (1)
      - 90-99 (2)
      - <90 (3)
    - other markers
      - HR >100 (1)
      - melaena (1)
      - syncope (2)
      - hepatic disease (2)

        - cardiac failure (2)
    - Rockall score
        - full score requires post endoscopy findings
        - pre-endoscopy score can be useful to predict those who will have high mortality and require critical care input
            - pre-endoscopy score of 3 has a mortality rate of around 10% and a score of 6 50%)
        - age
            - <60 (0)
            - 60-79 (1)
            - >80 (2)
        - shock
            - none, BP >100, HR <100 (0)
            - tachycardia, BP >100, HR 100 (1)
            - hypotension, BP <100 (2)
        - comorbidity
            - none (0)
            - cardiac failure or IHD (2)
            - renal failure, liver failure or disseminated malignancy (3)
        - endoscopy
            - no blood or dark spot only (0)
            - blood in upper GI tract, adherent clot or spurting vessel (2)
        - diagnosis
            - Mallory-Weiss tear (0)
            - all other diagnoses (1)
            - GI tract malignancy (2)
        - mortality
            - 0 – 0.2%
            - 1 – 2.4%
            - 2 – 5.6%
            - 3 – 11%
            - 4 – 24.6%
            - 5 – 39.6%
            - 6 – 48.9%
            - 7 – 50%
- management
    - non-variceal bleeds
        - PPIs
            - not currently recommended pre-endoscopy
            - they theoretically work by increasing gastric pH, thus promoting clot stability
        - somatostatins
            - octreotide is not recommended for acute non-variceal bleeds
            - can be used as an adjunct when there is a delay to endoscopy
        - antifibrinolytic therapy

        - should not be used – no benefit and slight increased risk of thrombus
- variceal bleeds
    - somatostatins and vasopressins
        - somatostatins (e.g. octreotide) cause relaxation of vascular smooth muscle and reduction of portal venous pressure
            - no reduction in deaths shown in systematic review
        - vasopressins (e.g. terlipressin) cause arterial vasoconstriction, reducing portal venous pressure but at the risk of end-organ ischaemia
            - shown to be safe and effective
            - reduces blood loss from actively bleeding varices and has a 34% relative risk reduction in risk of mortality
            - more convenient as can be given as a bolus
    - vitamin K
        - no evidence of efficacy in liver disease
    - antibiotics
        - 20% of cirrhotic patients with variceal bleeding develop a bacterial infection within 48 hours
        - all patients with suspected or confirmed varices should be given prophylactic antibiotics (e.g. ciprofloxacin, ceftriaxone)
- non-medical therapy
    - endoscopy
        - gold standard for diagnosis and treatment of UGIB
        - timing is important and should be after the patient is adequately resuscitated
        - common therapies include injection or thermal therapy for a bleeding peptic ulcer or banding of oesophageal varices
        - controls bleeding initially in around 90% of patients with bleeding peptic ulcers
    - balloon tamponade
        - Sengstaken-Blakemore tube (3 lumen) or Minnesota tube (4 lumen due to inclusion of oesophageal aspiration port)
        - used in a variceal bleed that continues despite medical therapy when endoscopy not immediately available
        - tamponade provides good control of bleeding in 90% - most will rebleed within 24 hours, and the procedure is just to buy time to endoscopy
        - procedure:
            - RSI to secure the airway
            - test the balloons on the tube to the maximally recommended volume then fully deflate them
            - lubricate the tube and pass orally to 50-60cm
            - insert air into the gastric aspiration port and listen with stethoscope over the stomach; inflate gastric balloon with 50ml air

                - confirm placement with CXR (gastric balloon in stomach)
                - inflate gastric balloon with air in 50-100ml increments up to the maximally recommended volume (250-300ml for SBT or 450-500ml for Minnesota); stop if resistance encountered; clamp the port
                - further x-ray to confirm position
                - pull balloon back against gastric fundus and mark at the lips; traction with 500ml bag of fluid and rope-and-pulley system
                - oesophageal balloon should only be inflated if bleeding continues (to 40mmHg)
            - complications include:
                - oesophageal necrosis and perforation from inflation of the oesophageal balloon, or the gastric balloon in an incorrect position
                - aspiration if the airway is not secured first
                - mucosal ulceration from pressure (should not be left in situ for more than 24-36 hours, and periodically deflated and reinflated)
                - proximal migration of the tube causing airway obstruction
                - ongoing haemorrhage from incorrect insertion or position
        - further treatments if endoscopy fails
            - surgery
            - angiography and embolization
            - TIPPS (transjugular intrahepatic porto-systemic shunt)
                - radiological intervention in which a connection is made between the portal and hepatic venous systems to reduce portal venous pressure
                - may be performed in patients with varices continuing to bleed despite other therapies
- lower gastrointestinal haemorrhage
    - background
        - accounts for around 20% of GI haemorrhages
        - usually causes less haemodynamic instability than upper GI bleed but can cause massive haemorrhage with shock
        - in the majority of cases the bleeding stops during the initial resuscitation
        - overall mortality rate is 2-4% (possibly up to 21% in those with massive haemorrhage)
        - definition
            - bleeding distal to the ligament of Treitz
            - passage of bright red blood per rectum (haemotochezia) without the presence of blood in gastric aspirate
    - clinical assessment
        - history

- nature of bleeding
    - frequency, colour, presence or absence of clots
- other symptoms
    - fatigue, chest pain, palpitations, shortness of breath
    - abdominal pain, weight loss, fever, diarrhoea, vomiting
- past medical history
- medication
    - particularly heparin, warfarin, NSAIDs, inhibitors of platelet aggregation

▪ examination

- full physical examination and observations
- any postural drop in blood pressure
- abdominal tenderness
- PR
    - stool colour, presence of blood, anorectal lesions

o risk assessment

▪ BLEED criteria (not well validated or in common use)

- ongoing bleeding
- hypotension (systolic <100)
- abnormal clotting (PT >1.2s)
- altered mental status
- significant comorbidities

▪ SIGN guidelines for admission

- consider for discharge or non-admission with outpatient follow-up if:
    - age <60 AND
    - no evidence of haemodynamic compromise AND
    - no evidence of gross rectal bleeding AND
    - an obvious anorectal source of bleeding on rectal examination/sigmoidoscopy
- consider for admission if:
    - age 60 or older OR
    - haemodynamic disturbance OR
    - evidence of gross rectal bleeding OR
    - taking aspirin or an NSAID OR
    - significant comorbidity

o differential

▪ diverticular disease

- commonest cause, involving colonic diverticula
- uncommon under 40

▪ inflammatory bowel disease

- including:
    - Crohn's disease
    - ulcerative colitis
    - ischaemic colitis
    - infective colitis
- ischaemic colitis is transient and reversible in most cases

        - the colon is predisposed to ischaemia due to poor collateral circulation, low blood flow and high bacterial content
        - the splenic flexure, recto-sigmoid junction and right colon are most commonly involved
        - more common in older adults due to presence of cardiovascular risk factors
        - tends to present with abdominal pain and bloody diarrhoea
    - neoplasia
        - carcinoma and polyps
        - present more commonly with occult bleeding than frank bleeding
        - incidence of colorectal carcinoma increases with age and family history
        - left sided large bowel carcinomas frequently cause altered bowel habit and abdominal pain
    - benign anorectal disease
        - rarely life-threatening but can be distressing
        - fissures are usually in the posterior midline if seen
        - patients with haemorrhoids tend to present with one of three complications: haemorrhage, prolapse or thrombosis
            - bleeding is usually self-limiting and rarely life-threatening
    - angiodysplasia
        - acquired malformation of the intestinal blood vessels with ectatic vessels in the mucosa and submucosa
        - dilated vessels or cherry red, flat lesions are seen at colonoscopy
        - approximately 80% are in the right side of the colon
        - most commonly presents with iron deficiency anaemia and occult blood loss
        - when overt bleeding occurs it is usually brisk, painless and intermittent
    - others
        - radiation injury
        - Meckel's diverticulum
        - other small bowel pathology
        - solitary rectal ulcers
        - portal colopathy
        - prostate biopsy sites
        - dieulafoy lesions
        - endometriosis
        - colonic varices
        - 10-15% of patients with an apparent lower GI bleed in fact have an upper GI bleed
- investigations may include
    - bloods
        - U&Es
        - FBC (Hb and platelets)
        - coagulation screen
        - LFTs

        - G&S or crossmatch
    - ECG
    - PR and faecal occult blood
    - CXR
    - VBG (or ABG)
    - nasogastric lavage or aspirate may be used to exclude upper GI bleed
- management
    - resuscitation as per UGIB
    - colonoscopy and treatment
        - low morbidity and mortality with yield of up to 86%
        - recommended initial investigation for most cases of LGIB
        - rapid bowel purge after initial resuscitation and colonoscopy within 24 hours of presentation
        - mucosa may be difficult to visualise during or soon after a bleed
        - coagulation can be performed during the procedure (thermal contact or epinephrine injection), particularly for AVMs
        - haemoclips and band ligation can also be done
        - there is a risk of perforation
    - angiography
        - thought to be preferable in patients with massive haemorrhage but success rates vary
        - source of bleeding is identified by visualising extravasation of contrast material into the bowel lumen
        - superselective embolization aims to decrease arterial blood flow which reduces pressure to the bleeding site to allow haemostasis
            - there is a small risk of ischaemia
        - NCEPOD recommends that hospitals that cannot provide onsite interventional radiology should have an agreed formalised local network
    - other investigative options:
        - proctoscopy
            - SIGN guidelines state that all patients with rectal bleeding should undergo proctoscopy
        - technetium labelled red blood cell scanning
        - upper GI endoscopy
        - CT angiography
        - recto-sigmoidoscopy
- medical management
    - treatment of patients taking warfarin as per local protocols
    - stop NSAIDs
    - low dose aspirin is normally continued
- surgical management
    - a small minority of patients require surgery
    - should be considered for a massive bleed or if other options have failed, taking into consideration extent and success of prior bleeding control measures, severity and source of bleeding and level of comorbid disease

        - if the bleeding point cannot be accurately detected, subtotal colectomy may be required
    - low risk bleeds
        - anal fissures
            - discharge with adequate analgesia and advice on diet/water intake or faecal softening agents
        - haemorrhoids
            - if otherwise stable, discharge home with faecal softening agents
            - may need further investigation to rule out more sinister cause for bleeding depending on age, other history and physical findings
            - investigations can often be performed as an outpatient

SuP6 anal/rectal pain

- functional anorectal pain syndromes
    - background
        - relatively common but consultation often delayed due to embarrassment and fear of sinister diagnosis
        - functional anorectal pain syndromes are defined by the Rome IV criteria
        - terms used are:
            - acute proctalgia – proctalgia fugax
            - levator ani syndrome
            - unspecified functional anorectal pain
        - the conditions are benign but can cause severe distress
    - aetiology
        - uncertain
        - proctalgia fugax is thought to occur due to spasm of the anal sphincter, levator ani syndrome due to spasm of the pelvic floor muscles, with the aetiology of unspecified functional anorectal pain unknown
            - there is overlap between them
        - they may be associated with irritable bowel syndrome
        - diagnosis can usually be made on the basis of symptoms and digital rectal examination, however more sinister causes can present in a similar way and it is important to exclude them
        - a history of anxiety/depression is often associated and should be explored
    - differential
        - irritable bowel syndrome
        - haemorrhoids +/- thrombosis
        - anal fissure
        - solitary chronic rectal ulcer
        - colorectal cancer
        - perirectal abscess or fistula; hidradenitis suppurativa
        - proctitis (especially gonococcal/chlamydial)
        - Crohn's disease/ulcerative colitis
        - rectal foreign body
        - pruritus ani
        - diverticular disease
        - rectal prolapse

        - coccygodynia (neuralgic pain around the region of the coccyx)
        - retrorectal cysts
        - conylomata acuminata (anogenital warts)
        - testicular tumours
        - prostatitis
        - interstitial cystitis
        - psychological cause
        - Alcock's canal syndrome (pudendal neuralgia due to entrapment)
        - hereditary anal sphincter myopathy
        - bilateral internal iliac artery occlusion
    - investigations
        - endoscopy (flexible rectosigmoidoscopy or colonoscopy) for chronic anorectal pain
        - other investigations if endoscopy normal and there is tenderness of the puborectalis muscle include:
            - anorectal manometry
            - balloon expulsion test
            - MR defecography
        - FBC, pelvic ultrasound and anorectal endosonography may also be considered
    - proctalgia fugax
        - symptoms
            - recurrent episodes of sudden, severe, cramping pain localised to the rectum
            - last from seconds up to 30 minutes and resolve completely
            - patient is entirely pain free between episodes
            - symptoms often occur at night and may wake the patient
            - attacks are infrequent (<5 times a year in 51%)
        - signs
            - there are no signs, diagnose is made on the basis of characteristic symptoms and the absence of other signs of pathology
            - abdominal and digital rectal examination are the minimum required
            - ideally, anoscopy/proctoscopy should be carried out
            - consider gynaecological/scrotal examination if indicated
            - some patients, where there is clinical suspicion of higher disease, may require sigmoidoscopy/colonoscopy
            - FBC to check for anaemia if suspicion of GI bleed
        - management
            - reassurance is usually sufficient once diagnosis made
            - drug therapy rarely required as symptoms are so transient
            - in patients with severe, frequent, prolonged attacks, inhaled salbutamol has been shown to reduce duration
            - other treatments are not supported by RCTs (e.g. sphincter relaxants such as oral diltiazem, topical GTN and nerve blocks
            - address psychological issues as needed
    - levator ani syndrome
        - symptoms

                - vague, aching or pressure sensation high in the rectum often worsened by sitting and relieved by walking
                - pain tends to be constant or recur regularly and to last >30 minutes
                - lasts from hours to days
                - for diagnosis, symptoms must be present for three months with symptom onset at least six months prior to diagnosis
                - other causes of similar pain must have been excluded
            - signs
                - posterior traction on the puborectalis reveals tight levator ani muscles and tenderness or pain (differentiates from unspecified functional anorectal pain)
                - tenderness may be predominantly left sided and massage of the puborectalis muscle may elicit the characteristic discomfort
            - management
                - patient education and reassurance
                - biofeedback or electrical stimulation
                - other treatments that have shown some benefit include:
                    - digital massage
                    - muscle relaxants
                    - sitz baths (shallow bath of lukewarm water)
    - unspecified functional anorectal pain
        - patients tend not to respond to biofeedback
        - biofeedback responsive patients can often be identified by a balloon evacuation test using a Foley catheter
        - addressing depression and anxiety may be useful
        - botulinum toxin injections may relieve symptoms
- rectal examination
    - anatomy
        - the rectum is the curved, lower, terminal segment of large bowel
        - it is about 12cm long and runs along the concavity of the sacrum
        - the upper two thirds is covered by peritoneum but the posterior rectum is not
            - in men, the anterior rectal peritoneum reflects onto the surface of the bladder base
            - in women, the anterior rectal peritoneum forms the rectouterine pouch (pouch of Douglas) – the pouch of Douglas is filled with loops of bowel
        - anterior to the lower one third of the rectum lie different structures in men and women
            - in men: the prostate, bladder base and seminal vesicles
            - in women: the vagina – it may be possible to feel cervix and even a retroverted uterus with the tip of the examining finger
        - the anus is 3-4cm long and joins the rectum to the perineum
        - the wall of the anus and anal canal is supported by powerful sphincter muscles made up of:
            - voluntary external sphincter muscles
            - involuntary internal sphincter muscles

        - these muscles are essential in the mechanism of defecation and the maintenance of continence
    - indications for rectal examination include:
        - assessment of prostate
        - when there has been rectal bleeding
        - constipation
        - change of bowel habit
        - problems with urinary or faecal continence
        - rarely to detect uterus and cervix when vaginal examination not possible
    - procedure
        - consent and chaperone
        - patient in left lateral position with flexed hips and knees and buttocks at the edge of the couch
        - gently part the buttocks to expose the anal verge and natal cleft
        - inspection of skin and anal margin with good light
        - lubricate the index finger and press against the posterior anal margin (6 o'clock)
            - finger should slip easily into the anal canal and the fingertip is directed posteriorly following the sacral curve
            - if appropriate, anal tone can be checked by asking the patient to squeeze the finger with the anal muscles
        - the finger is then moved through 180 degrees, feeling the walls of the rectum
            - with the finger in the 12 o'clock position, usually with the examiner in a half-crouched position and the examining wrist pronated, the anterior wall can be palpated
            - the opposing walls can then be palpated with rotation
        - in men, the prostate gland will be felt anteriorly
        - in women, the cervix and a retroverted uterus may be felt with a fingertip
        - it is important to feel through a full 360 degrees so as not to miss any small lesions
        - examination of the prostate gland:
            - normal size is 3.5cm wide, protruding about 1cm into the lumen of the rectum
            - normally rubbery and firm with a smooth surface and a palpable sulcus between right and left lobes
            - there should not be any tenderness or nodularity
            - massage of the prostate gland may enable prostatic fluid to be examined at the urethral meatus
        - on removal of the finger check the tip of the glove (stool, blood)
    - external inspection findings
        - skin disease
        - skin tags
        - pilonidal sinus
        - genital warts
        - anal fissures
        - anal fistula

        - external haemorrhoids
        - rectal prolapse
        - skin discolouration with Crohn's disease
        - external thrombosed piles
    - internal examination findings
        - simple piles (best examined at proctoscopy)
        - rectal carcinoma
        - rectal polyps
        - tenderness (for example in acute appendicitis)
        - diseases of the prostate gland
        - malignant or inflammatory conditions of the peritoneum (felt anteriorly)

SuP7 nausea/vomiting

- persistent nausea/vomiting
    - background
        - there are varied diagnoses under five main headings:
            - pregnancy
            - visceral disease
            - toxic substance effects/metabolic conditions
            - central nervous system disease
            - psychiatric illness
    - assessment
        - assessment of the physical state of the patient
            - poor nutritional state
            - dehydration
            - electrolyte imbalance
        - assessment with regard to the underlying cause
    - pregnancy
        - nausea and vomiting affects up to 75% of pregnant women
        - around 1% develop hyperemesis gravidarum
            - may result in adverse outcomes for mother and foetus
        - other causes:
            - morning sickness
            - UTI
            - reflux oesophagitis
            - mechanical pressure from the gravid uterus
    - visceral disease
        - reflux oesophagitis or GORD
        - obstruction (e.g. due to malignancy or chronic constipation)
        - cholecystitis
        - hepatitis
        - UTI
        - gastroparesis (delayed gastric emptying)
    - toxic substance effects/metabolic conditions
        - drugs
            - e.g. cytotoxic agents, erythromycin, digoxin toxicity, theophylline
        - alcohol

        - hypercalcaemia
        - uraemia
        - diabetic ketoacidosis
    - central nervous system disease
        - cyclical vomiting syndrome
            - recurrent discrete episodes of vomiting in an otherwise healthy person, usually a child
            - may have abdominal pain and family history of migraine
        - vestibular labyrinthitis and Ménière's disease
        - raised intracranial pressure
    - psychiatric disease
        - bulimia nervosa
        - functional
        - psychogenic
    - common causes of persistent nausea and vomiting and their mechanisms
        - irritation or stretching of the meninges
            - examples:
                - raised intracranial pressure caused by intracranial tumour
            - mechanisms:
                - unknown, may involve meningeal mechanoreceptors
        - pelvic or abdominal tumour
            - examples:
                - mesenteric metastases
                - metastases of liver
                - ureteric obstruction
                - retroperitoneal cancer
            - mechanisms:
                - stretching of mechanoreceptors
        - bowel obstruction secondary to malignancy
            - examples:
                - mechanical
                    - intrinsic or extrinsic by tumour
                - functional
                    - disorders of intestinal motility secondary to malignant involvement of nerves, bowel muscle or blood supply
                - paraneoplastic neuropathy
            - mechanisms:
                - stretching of mechanoreceptors
        - gastric status
            - examples:
                - drugs (anticholinergics, opioids)
                - mechanical obstruction to gastric emptying – tumour, gastritis, peptic ulcer, hepatomegaly
                - autonomic failure (e.g. advanced diabetes)
            - mechanisms:
                - gastric mechanoreceptors

        - chemical/metabolic
            - examples:
                - drugs (anti-epileptics, opioids, antibiotics, cytotoxics, digoxin)
                - metabolic (e.g. hypercalcaemia)
                - toxins (e.g. tumour necrosis, bacterial toxins)
            - mechanisms:
                - chemoreceptors in the trigger zone
        - anxiety induced
            - examples:
                - concern about diagnosis, treatment, symptomatology, social issues, anticipatory emesis with cytotoxics
            - mechanisms:
                - multiple receptors in the cerebral cortex
        - movement related
            - examples:
                - abdominal tumours
                - opioids
                - disease affecting the vestibular system
            - mechanisms:
                - accentuation of stretch of mechanoreceptors by tumours
                - vestibular sensitivity increased by opioids
                - vestibular function disturbed by pathology
    - history
        - duration
        - severity
        - aggravating and relieving factors
        - associated features
        - drug history
        - occupational history
        - social history
        - last menstrual period
        - previous medical history
        - recent trauma
    - examination
        - including assessment of hydration and nutritional state, examination of abdomen, sclera, optic discs, check for nystagmus
    - investigations may include:
        - urine dipstick
        - U&Es
        - calcium
        - LFTs
        - FBC
        - pregnancy test
        - abdominal x-ray
        - abdominal ultrasound
        - endoscopy

        - abdominal CT/MRI
        - CT/MRI of brain if raised intracranial pressure suspected
    - management
        - general measures
            - dietary advice and advice on fluid intake
            - IV fluids if severe dehydration
            - psychiatric or psychology input if psychological cause identified
            - pregnant women:
                - emotional support
                - advice on diet and adequate nutritional intake
                - avoidance of large volume meals
                - avoidance of tight clothing
            - acupuncture may be helpful for some patients
        - pharmacological
            - various classes of drug have antiemetic properties (e.g. antihistamines, phenothiazines such as prochlorperazine and antipsychotics such as haloperidol
            - metoclopramide
                - acts directly on the GI tract
                - risk of potentially serious adverse neurological effects such as extrapyramidal effects and tardive dyskinesia
                - should only be prescribed for up to 5 days
            - cyclizine and metoclopramide are safe and effective in pregnancy
            - domperidone acts at the chemoreceptor trigger zone and is especially useful for chemotherapy-related nausea and vomiting
                - it is also prokinetic and can be useful for gastroparesis
            - gastro-electrical stimulation is also an option for chronic, intractable nausea and vomiting related to gastroparesis
            - granisetron and ondansetron are specific 5HT3 receptor antagonists and are particularly useful for post-operative or cytotoxic therapy related nausea and vomiting, and in children
            - dexamethasone and nabilone (synthetic cannabinoid) can be useful for patients on cytotoxic drugs with nausea resistant to other therapies

    - complications of recurrent vomiting
        - dehydration
        - electrolyte disturbance
        - oesophagitis/gastritis
        - Mallory-Weiss syndrome
- cannabinoid hyperemesis syndrome
    - background
        - cyclical vomiting associated with abdominal pain associated with long-term or excessive cannabis use
        - patients often compulsively shower to provide transient relief
    - theories for pathophysiology

        - susceptible patients may develop a hypersensitivity to cannabis following years of exposure
        - cannabis has a long half-life of weeks or months in the body; regular use leads to accumulation which can give rise to hypersensitivity in susceptible patients
        - cannabis delays gastric emptying which may lead to gastric stasis and therefore hyperemesis
        - compulsive bathing due to the presence of cannabinoid receptors in the limbic system of the brain; the toxicity may disrupt the thermoregulatory systems of the hypothalamus, which may settle with bathing/hot shower
    - diagnosis
        - features include:
            - history of several years of cannabis use prior to onset
            - cyclical pattern every few weeks or months, often for many years, against a background of regular cannabis use
            - cessation of cannabis leads to cessation of hyperemesis in the presence of a negative urine drug screen for cannabinoids
            - a return to cannabis use leads to return of hyperemesis weeks or months later
            - the patient compulsively bathes
    - differential of cyclical vomiting
        - hyperemesis gravidarum
        - metabolic disorders (e.g. Addison's, porphyria)
        - paediatric cyclical vomiting
        - migraine variants
        - drug withdrawal syndrome
        - bulimia and anorexia nervosa
    - investigations
        - patient may have been seen multiple times and have had previous investigations
        - investigations may include:
            - blood glucose
            - VBG for acid-base status
            - urinalysis including beta hCG
            - FBC, U&E, LFT, amylase
            - drug screen if available and suspicion of use despite patient denial (cannabinoids can be detected up to 6 weeks after cessation of chronic use)
    - management
        - cannabis withdrawal chart (if available)
        - IV hydration if dehydrated
        - correct electrolyte imbalance (especially potassium and magnesium)
        - antiemetics, e.g.:
            - metoclopramide 10-20mg IV
            - ondansetron 4-8mg IV
            - prochlorperazine 12.5mg IV
        - consider antispasmodic (buscopan 10-20mg IV)

        - if nausea and vomiting persist, consider:
            - antipsychotics and low dose benzodiazepines (e.g. midazolam 0.5-1mg IV boluses titrated to effect)
                - have antiemetic effects and help relieve agitation
        - long term management
            - abstinence is the definitive treatment
            - follow up by drug and alcohol services

SuC1 ano-rectal abscesses

- anatomy
    - anorectum formed by the 12-15cm rectum and the 4cm anal canal
    - columnar intestinal epithelium of the rectum changes to stratified squamous epithelium at the dentate or pectinate line
        - this is where the anal glands and crypts lie
    - the anorectum is supplied by the haemorrhoidal arteries and drained by the internal haemorrhoidal plexus of veins
    - sphincteric support is provided by the internal sphincter, puborectis muscle and external sphincter
        - the external sphincter has three parts:
            - deep
            - superficial
            - subcutaneous
        - it is a continuation of the puborectalis muscle
        - they form a ring of muscle in the anus with an important function in the maintenance of continence
        - several tissue spaces lie between the muscles and are sites of potential abscess and fistula formation
- history
    - systemic symptoms
        - weight loss
        - fever
        - can suggest systemic disease with anorectal manifestations such as inflammatory bowel disease
    - recurrence
        - can indicate an underlying disease such as diabetes, carcinoma or immunosuppression
        - multiple fistulas and fistula formation suggest an underlying systemic cause
    - altered bowel habit
    - sexual history
        - sexually transmitted infections with anorectal manifestations include chlamydia, herpes, syphilis, condylomata acuminata (anogenital warts)
    - age
        - patients over 40 with rectal bleeding should be investigated for possible malignancy
- examination
    - pain

        - a patient with rectal pain and no apparent abnormality may have a serious deep abscess
    - PR
        - inspection
            - well-lit area with patient in left lateral position
            - look for obvious abscesses or areas of induration or pain suggesting deeper abscess
            - note external haemorrhoids and appearance
                - purplish tender grape-like haemorrhoids tend to be thrombosed
                - black haemorrhoids tend to be necrotic
            - anal fissures
            - look in the natal cleft for pilonidal sinuses
        - digital rectal examination
            - often normal with haemorrhoids
            - often painful and poorly tolerated with anal fissure or abscess
        - proctoscopy/anoscopy must be performed
            - may be by the surgical team in the ED or as an outpatient
- anal fissure
    - superficial tears of the anal mucosa
        - occurs when passing a hard stool
        - normally in the posterior midline (point of maximum tissue weakness)
        - a sentinel pile or skin tag may occur at the distal end of the fissure
        - cause severe pain on defecation lasting for 1-2 hours afterwards
        - tend to cause small amount of bright red blood on paper
        - management:
            - discharge home with analgesia, stool softeners and surgical follow up
            - pain is usually due to spasm of the internal sphincter rather than the fissure
                - theoretically anything that can reduce spasm will promote healing
                - GTN ointment usually used first line (trials show it does not improve healing rate
            - surgical:
                - sphincterotomy effective in 90%
                    - 10% of patients will develop long term incontinence
                    - anal stretch results in higher rates of incontinence
- anorectal abscesses
    - can affect various areas of the anorectum
    - can be defined by anatomic location
    - the anal crypts can become blocked leading to abscess formation
        - Staph aureus is the commonest cause
    - chronic infection can lead to the development of a fistula
    - locations:
        - perianal (40%)
            - pain and fluctuant swelling at anal verge

        - ischiorectal (20%)
            - buttock pain and induration, may be no swelling
        - intersphincteric (20%)
            - rectal fullness on PR
        - supralevator (5%)
            - buttock and rectal pain, little external evidence
    - management:
        - all require surgical intervention
        - no role for treatment of closed abscess with antibiotics
            - adjunctive antibiotics only required if the abscess is complex, deep, associated with cellulitis or the patient is immunosuppressed
        - incision and drainage required to reduce risk of sepsis and fistula formation
            - usually done under general anaesthesia in theatre
            - some superficial abscesses are drained in the ED (more common in the US)
- pilonidal disease
    - spectrum of conditions ranging from pilonidal sinuses in the natal cleft to the formation of a laterally placed pilonidal abscess
    - a pilonidal sinus can occur when there is blockage of a hair follicle in the natal cleft
        - the sinus can become infected leading to chronic sinus inflammation or formation of a pilonidal abscess laterally to the sinus
    - pain worse on defecation or sitting
    - tender swelling
    - deep pain or buttock pain depending on location
    - small amounts of blood or pus may occur PR if fistula present
    - management:
        - if no abscess present, good hygiene and shaving of hairs in the natal cleft promote healing
        - surgical management of abscess
            - incised off midline
            - sinuses should be fully incised once the abscess has settled
            - pilonidal sinuses and anorectal fissures heal best with primary closure after excision
- rectal prolapse
    - protrusion of part or all of the rectal canal
    - largely painless unless strangulated
    - blood stained mucus PR
    - management:
        - refer to surgeons
            - excision and pelvic floor repair

SuC2 appendicitis

- background
    - most common between ages of 15 and 25
    - affects around 10% of people
    - more common in men
- anatomy

    - innervated by the autonomic nervous supply to the mid gut
    - inflammation in the appendix activates afferent sympathetic fibres
        - these enter the spinal cord at T10 resulting in referred pain to the periumbilical area (visceral pain)
    - inflammation later irritates the surrounding parietal peritoneum
        - this is innervated by the intercostal nerves and results in constant RIF pain (somatic pain)
- differential
    - gastrointestinal
        - terminal ileitis
        - mesenteric adenitis
        - Meckel's diverticulitis
        - diverticulitis
        - acute cholecystitis
        - gastroenteritis
        - bowel obstruction
        - non-specific abdominal pain
    - gynaecological
        - ruptured ovarian cyst
        - ectopic pregnancy
        - pelvic inflammatory disease
        - ovarian torsion
    - urological
        - renal colic
        - urinary tract infection
        - pyelonephritis
- history
    - classical presentation (50% of patients)
        - initial central abdominal colicky pain
        - moves to the right iliac fossa after 6-12 hours and becomes constant
    - other common symptoms:
        - anorexia (80% of patients)
        - nausea
        - vomiting (starts after the pain)
        - constipation
        - diarrhoea
    - often atypical in the elderly, children and pregnant patients
        - may result in late diagnosis and high risk of perforation
- examination
    - may look flushed and dehydrated
    - may have a furred dry tongue and bad breath
    - may have a fever and slight tachycardia
        - patients with a perforated appendix are more likely to have a high temperature
    - abdominal examination
        - patients are in pain and want to lie still
        - may have RIF tenderness, maximal at McBurney's point

                - one third of the way between the anterior superior iliac spine and the umbilicus
        - signs of localised peritonism
            - direct tenderness
                - patient has pain on palpation of RIF
            - guarding
                - due to voluntary or involuntary contraction of abdominal muscles
            - rigidity
                - due to reflex spasm of abdominal wall muscles
            - rebound
                - pressure for 30 seconds in the RIF, patient has pain when hand removed
            - Rovsing's sign
                - pressure for 30 seconds in the LIF, patient experiences pain in the RIF when released
                - due to stretching of the whole peritoneum
            - psoas sign
                - patient lifts flexed thigh against hand placed above knee, patient experiences pain in the RIF
            - kinder ways to elicit signs of localised peritonism:
                - ask patient to suck in and push out their abdomen
                - ask patient to cough gently
                - gentle percussion over the RIF to elicit guarding and rigidity
- risk assessment
    - no tools predict morbidity and mortality
- investigation
    - urinalysis
        - up to 30% of patients with appendicitis have abnormal results
        - consider urgent Gram stain if in doubt
    - urinary beta hCG
        - exclude ectopic in any female of child bearing age
    - FBC
        - 80-90% have a raised WCC
        - does not rule in or out appendicitis
    - CRP
        - has specificity/sensitivity around 50-60%
- clinical scoring tool
    - Alvarado score
        - validated across all patient groups
        - sensitivity of around 72%, should be used alongside clinical judgement
        - score of 5-6 considered suggestive of appendicitis
            - symptoms
                - abdominal pain that migrates to the right iliac fossa (1)
                - anorexia or urinary ketones (1)
                - nausea or vomiting (1)
                - tenderness in the right iliac fossa (2)

        - signs
            - rebound tenderness (1)
            - fever of 37.3 or more (1)
        - laboratory
            - leucocytosis >10,000 (2)
            - neutrophilia (>70%) (1)
- imaging
    - US
        - graded compression US has an overall accuracy of about 90% but very operator dependent
        - can rule in appendicitis but not rule it out
        - has value in trying to identify other pathologies
    - CT
        - accuracy around 94%
        - can rule in but not out
- diagnostic difficulties
    - children
        - atypical symptoms and signs can lead to late presentation
    - elderly
        - atypical symptoms and signs can lead to late presentation
        - 3x increased rate of perforation
    - pregnant patients
        - abnormal position of the appendix due to the uterus can cause atypical signs
        - perforation is associated with foetal mortality
    - abnormal positioning of the appendix
        - atypical site of pain
    - women of child-bearing age
        - extensive differential including tubo-ovarian pathologies
        - higher rates of negative appendicectomies
- management
    - low risk patients
        - may be discharged home with advice to return if their symptoms recur or change
    - resuscitation
        - if dehydrated or signs of sepsis
        - oxygen
        - IV fluids
        - antibiotics if signs of septicaemia or generalised peritonitis
            - should also be given if decision made to take patient to theatre
    - analgesia
        - morphine with anti-emetic
    - keep nil by mouth
    - surgical review
        - if diagnosis not clear, may be admitted for serial examinations, US or CT
- surgical approach
    - usually laparoscopic appendicectomy

- diagnostic laparoscopy may be used when diagnosis remains uncertain, in conjunction with gynaecology team if indicated
- isolated appendiceal masses or abscesses without risk of perforation may be treated conservatively with IV antibiotics

SuC3 biliary colic

- background
  - gallbladder disease is the commonest abdominal complaint
  - 2-3 times more common in females
    - likely due to oestrogen moving cholesterol from blood to liver
  - obesity is the strongest independent risk factor in women
- pathophysiology
  - gallstones are precipitates of bile that form in the gallbladder
  - 50% of bile is released into the duodenum when fasting, 50% stored in the gallbladder
  - normal bile:
    - 70% bile salts
    - 22% phospholipids
    - 4% cholesterol
    - 3% proteins
    - 0.3% bilirubin
  - stone types
    - white/mixed
      - pure or mainly cholesterol
      - 80% of UK gallstones occur when bile is supersaturated with cholesterol
    - brown
      - <5% in UK
      - due to stasis and infection within the biliary tree
    - black
      - bile predominant
      - seen in haemolytic disease (sickle cell, thalassaemia, hereditary spherocytosis etc) and cirrhosis
- risk factors for gallstones
  - increasing age
  - female sex
  - familial predisposition
  - pregnancy
  - hyperlipidaemia
  - bile salt loss (ileal disease, resection, clofibrate drugs)
  - diabetes
  - cystic fibrosis
  - total parenteral nutrition
  - prolonged fasting
- types of gallstone disease
  - asymptomatic gallstones
    - gallstone passes freely through biliary tract to gut

    - biliary colic/chronic cholecystitis
        - stone gets intermittently stuck in biliary tract
    - gallstone stuck for prolonged period
        - results in a number of conditions:
            - acute cholecystitis
            - ascending cholangitis
            - acute pancreatitis
            - gallbladder perforation
            - empyema
    - gallstone ileus
        - occurs when the stone gets stuck outside the biliary tract
- examination
    - patient may be flushed, tachycardic, jaundiced, dehydrated, hypotensive
    - may have epigastric or RUQ tenderness or diffuse abdominal tenderness
    - Murphy's sign – when the examiner presses deeply in the right subcostal region, with a sharp increase in pain as the inflamed gallbladder touches the examiner's hand
        - can cause inspiratory arrest
        - around 97% sensitive for acute cholecystitis
    - isolated rebound tenderness in the RUQ
        - reflects localised peritoneal irritation secondary to leakage of inflammatory fluid or gallbladder contents
        - generalised rigidity and peritonism is rare
            - reflects leakage of gallbladder contents into the peritoneal cavity
    - jaundice
        - rarely found
        - represents chronic obstruction, chronic intravascular haemolysis or development of the Charcot triad (fever, RUQ pain and jaundice – only seen in 25% of cases of ascending cholangitis)
- investigation
    - urinalysis
        - unconjugated bilirubin in haemolysis will not appear in the urine (tightly bound to albumin)
        - conjugated bilirubin is water soluble so will appear in the urine
        - urobilinogen (bilirubin breakdown product excreted in the blood) does not appear in the urine in cholestasis
    - urinary beta hCG in women of child-bearing age
    - bloods
        - FBC
        - U&Es
        - ALP
            - may be raised
            - marker for biliary tract obstruction but non-specific
                - has isoenzymes in liver, bone,, placenta, small bowel and white cells
            - may not be immediately raised in acute obstruction
        - ALT and bilirubin

        - may also be normal
    - amylase
        - to exclude pancreatitis
- imaging
    - x-ray
        - only 10% of gallstones are visible on plain films
    - US
        - can pick up gallstones within the gallbladder of 1-2mm diameter with 95% sensitivity and 97% specificity
        - may miss 25-40% of bile duct stones due to overlying bowel gas
        - acute cholecystitis shows a thickened gallbladder wall, pericholecystic fluid and a sonographic Murphy's sign
    - CT
        - not generally helpful to diagnose acute cholecystitis
- management
    - consider discharge from ED if an acute attack of biliary colic has settled
    - resuscitation
        - if dehydrated or signs of sepsis
        - may need critical care involvement
    - analgesia
        - opiates as needed
    - antibiotics
        - IV broad spectrum antibiotics if signs of sepsis, acute cholecystitis or complicated gallbladder disease
    - keep nil by mouth
    - medical management
        - not commenced in the ED
        - may be appropriate for certain patients with high surgical risk
            - oral bile salt therapy can prevent development of gallstones in high risk groups but is not useful once they have developed
                - ursodeoxycholic acid is used
        - extracorporeal shockwave lithotripsy may be used with sphincterotomy and a percutaneous approach
            - high failure rate when used alone (95%), high complication rate (19%)
    - surgical management
        - not indicated in asymptomatic disease except in some select groups (e.g. sickle cell disease)
        - laparoscopic versus open cholecystectomy shows no difference between mortality, operating time or complication rate but there is an extension of hospital stay with the open procedure
        - ERCP (endoscopic retrograde cholangiopancreatography) in patients with gallstone pancreatitis

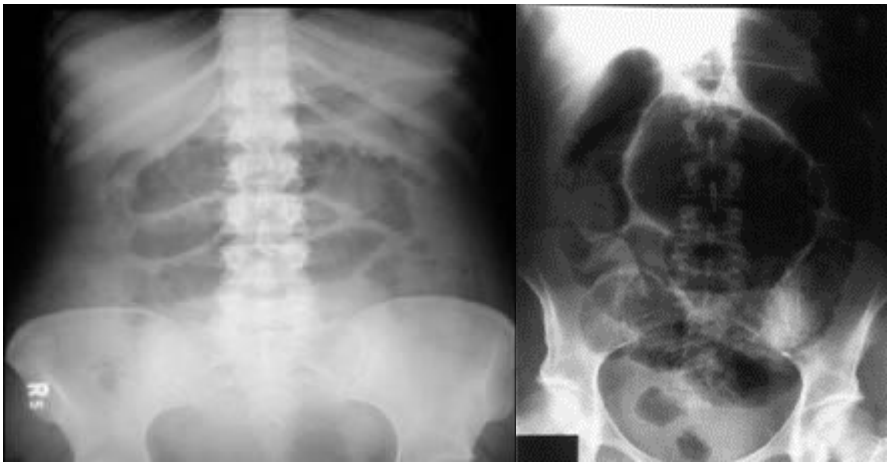
SuC4 bowel obstruction

- background
    - 5% of emergency surgical admissions

    - mortality varies widely by cause
    - classifications:
        - small/large bowel
        - acute/ subacute/ acute on chronic/ chronic
        - mechanical/functional
        - simple/strangulated
        - partial/complete
    - commonest causes:
        - small bowel adhesions (60% in UK), hernias, intussusception (paeds)
        - large bowel malignancy (developed countries), volvulus (low resource countries)
        - functional causes are relatively rare as ED presentations
- anatomy and pathophysiology
    - small bowel extends from the pylorus to the ileocaecal valve
        - length from 3 to 9 metres
        - diameter of lumen narrows progressively
    - large bowel extends from ileocaecal valve to anus and rectum
        - if the ileocaecal valve remains competent in colonic obstruction, perforation may result from increasing pressure
    - innervation
        - via the parasympathetic and sympathetic nervous systems which form the myenteric plexus
            - controls orderly propulsive contractions of the muscular layer of the gut wall
        - the sympathetic nervous system mediates the sensation of visceral pain
            - poorly localised, periumbilical and colicky
            - when the gut contracts against an object
    - universal findings of intestinal obstruction
        - sequestration of fluids and gas proximal to the obstruction
        - dilatation of bowel and abdominal distension
        - fluids shift out of the bowel into the peritoneum +/- perforation
            - fluid losses are generally greater the more proximal the obstruction
                - fluids secreted by the upper GI tract fail to reach the absorptive areas of the distal jejunum and ileum
        - irritation of peritoneum
        - vascular shifts into the peritoneal cavity
        - hypovolaemia, dehydration and shock
            - third space losses can be large and underestimated
- aetiology
    - mechanical
        - intrinsic (intramural)
            - congenital atresia
            - inflammatory strictures
                - Crohn's disease
                - TB
                - diverticular disease
            - tumours

                        - benign
                        - malignant
                    - radiation stricture
                    - ischaemic stricture
                - extrinsic (extramural)
                    - adhesions
                    - hernias
                    - volvulus
                    - intussusception
                    - congenital bands
                    - inflammatory masses
                    - tumours
                        - benign
                        - malignant
                - luminal (intraluminal)
                    - constipation
                    - foreign bodies
                    - gallstones
                    - parasites
                    - bezoars
            - functional
                - results from atony of the intestine and loss of normal peristalsis
                - systemic
                    - hypokalaemia
                    - hyponatraemia
                    - hypothermia
                    - hypoxia
                    - DKA
                    - uraemia
                    - drugs (e.g. anticholinergics)
                    - general anaesthesia
                    - dehydration
                    - sepsis
                    - retroperitoneal malignancy
                    - trauma
                        - head injury, spinal injury, pelvic surgery
                - local
                    - infection
                        - intra-abdominal infection/peritonitis
                        - strongyloides
                    - trauma
                    - postoperative (ileus)
                    - vascular (mesenteric ischaemia)
- history
    - classical symptoms
        - colicky abdominal pain

        - abdominal distension
            - may not be significant with high small bowel obstruction
            - may be gross distension with low small bowel or large bowel obstruction
        - vomiting
            - late feature with large bowel obstruction
        - constipation
    - history features
        - absence of passage of faeces or flatus
        - previous abdominal surgery
        - reproductive, contraceptive and menstrual history
        - possibility of pregnancy and its complications
        - drug history
        - alcohol history
        - past medical history
        - severe pain
            - suggests strangulation and developing ischaemia in a closed loop of bowel
- examination
    - surgical scars
    - peritonism
    - masses
    - hernial orifices
        - it is easy to miss a small femoral hernia in an obese patient
    - bowel sounds
        - may be high pitched/tinkling or absent
    - rectal examination
        - may be empty rectum or obstructing mass
        - ideally should only be performed once
- investigations
    - general
        - FBC, U&E, LFT, amylase, glucose, G&S, VBG
        - coagulation screen if septic or on anticoagulants
        - erect chest x-ray and supine abdominal x-ray
            - small bowel distension >2.5cm
                - valvulae conniventes completely cross the bowel wall and confirm small bowel
                - taeniae coli of large bowel are incomplete across the bowel wall



small bowel and large bowel obstruction

https://www.rcemlearning.co.uk/reference/bowel-obstruction/#1568203211329-68736ad1-f52c

        - ECG
        - urinalysis
    - imaging
        - CT
            - useful for differentiating specific cause and location
            - has low sensitivity for detecting low-grade or partial small bowel obstruction
        - small bowel follow through
        - water-soluble contrast enema
- ED management
    - IV fluids
    - oxygen as needed
    - morphine and antiemetic
    - NG tube and nil by mouth
    - urinary catheter
    - invasive monitoring if required
    - consider broad spectrum antibiotics if signs of strangulation or peritonism
- surgical management
    - usually period of non-operative observation unless patient unwell
    - further imaging including contrast studies
    - colonoscopy may be done for likely large bowel obstruction
        - procedure may relieve pseudo-obstruction
    - large bowel obstruction generally requires operative intervention
        - usually primary resection and anastamosis

SuC5 breast abscess

- mastitis background
    - may be infective or non-infective
        - non-infectious
            - accumulation of milk causes an inflammatory response in the breast
        - infectious
            - accumulated milk allows bacteria to grow

        - usually Staph aureus, may be Staph albus or Strep
    - 10-33% of breastfeeding women develop lactation mastitis
    - incidence is highest in the first few weeks postpartum
        - may occur as long as the woman is breastfeeding
    - may develop into breast abscess
- risk factors
    - problems with attachment of infant to breast during feeding
        - problems with technique
        - anatomical anomalies such as tongue tie or cleft lip
    - reduced number or duration or feeds leading to milk accumulation
        - partial bottle feeding
        - changes in regime (e.g. infant sleeping through night)
        - rapid weaning
        - painful breasts
        - preferred breast, leading to milk accumulation in the other
    - pressure on the breast
        - tight clothing
        - seat belt
        - sleeping in prone position
    - nipple fissures, cracks and sores
    - trauma to breasts
    - blocked milk ducts
- symptoms
    - normally presents ≥1 week postpartum
    - usually unilateral
    - painful, tender, hot, red affected area
    - systemic symptoms
        - fever
        - rigors
        - muscle pain
        - lethargy
        - depression
        - nausea
        - headache
    - should be distinguished from congestive mastitis (breast engorgement) which usually presents on second or third day of breastfeeding
        - presents as swollen and tender breasts bilaterally without fever or erythema
- signs
    - unilateral oedema
    - erythema in wedge-shaped area
    - tenderness
    - area feels firm and hot
    - fever may be present
    - not possible to distinguish clinically between infectious and non-infectious mastitis
    - if abscess has developed there will be a fluctuant tender lump with overlying erythema
    - may be axillary lymphadenopathy palpable
- investigations

    - diagnosis is usually clinical
    - milk should be cultured if infection is severe or recurrent or not starting to resolve after 2 days of antibiotics
        - also if infection acquired in hospital
    - ultrasound for abscess if infection not settling after one course of antibiotics
- management
    - reassurance
        - mastitis is painful but should not interfere with ability to breastfeed or long term appearance of breast
    - encourage continued breastfeeding
        - consider expressing if too painful to breastfeed
    - improve milk removal
        - assessment of breastfeeding technique by skilled person
        - manual expression of milk to empty breast after feeding
        - self-massage of the breast before feeding or expression or application of heat by warm compresses, shower or heat packs
        - increased feeding frequency
        - feeding on the affected side first while symptoms persist so the breast is emptied most effectively
    - analgesia
        - paracetamol or ibuprofen
    - not wearing a bra at night
    - emotional support
    - antibiotics
        - should not be a first line treatment
        - start after 24 hours of first line measures if not improving
        - start immediately if acutely unwell or infected nipple injury present
        - usually flucloxacillin or erythromycin
    - surgical management
        - indicated for breast abscess
            - incision and drainage with the cavity packed open with gauze if the overlying skin is thin or necrotic
        - parenteral antibiotics with coverage for anaerobes
        - culture of fluid from abscess
        - an alternative may be needle aspiration repeated every other day until pus no longer accumulates
        - breast feeding may have to cease until the abscess is successfully treated but can usually resume later
        - any persisting mass will need further investigation
- when to suspect an abscess
    - history of recent mastitis or prior breast abscess
    - fever and/or general malaise
        - may have settled if she has taken antibiotics for mastitis
    - painful swollen lump in the breast with redness, heat and swelling of the overlying skin
        - lump may be fluctuant with skin discolouration
    - risk factors for development of abscess include:
        - sudden cessation of breastfeeding in mastitis

        - obesity
        - smoking

SuC6 cholangitis

- background
    - up to 9% of patients admitted to hospital with gallbladder disease have acute cholangitis
    - equal male to female ratio
    - median age of presentation 50-60 years
    - malignant disease accounts for 10-30% of cases
- causes
    - obstruction of the gallbladder or bile duct due to stones
    - ERCP
    - tumours
        - pancreatic cancer
        - cholangiocarcinoma
        - ampullary cancer
        - porta hepatis tumours
        - metastasis
    - bile duct stricture or stenosis
    - choledochocoele (cyst or diverticulum of the CBD)
    - AIDS cholangiopathy
    - parasitic infection
        - roundworm
        - liver fluke
- presentation
    - Charcot's triad (jaundice, fever, RUQ pain) in 50-70%
        - 10-20% also have hypotension due to septic shock and confusion
    - abdominal pain may be poorly localised in elderly patients
    - may have acholic (putty coloured) stools and pruritus
    - history of gallstones, CBD stones, recent cholecystectomy, ERCP or other invasive procedures, HIV or AIDS
- signs
    - fever
    - right upper quadrant tenderness
    - jaundice
    - mental status changes
    - hypotension
    - tachycardia
    - peritonism is unusual and should prompt a search for an alternative diagnosis
- grading of acute cholangitis
    - grade I (mild)
    - grade II (moderate)
    - grade III (severe)
        - associated with at least one of cardiovascular, neurological, respiratory, renal, hepatic and/or haematological dysfunction
- diagnostic criteria

    - A: systemic inflammation
        - A1: fever and/or shaking/chills, temperature >38°
        - A2: evidence of inflammatory response – abnormal WCC, increased CRP, other changes indicating inflammation
    - B: cholestasis
        - B1: jaundice
        - B2: abnormal LFTs – increased ALP, GGT, AST, ALT
    - C: imaging
        - C1: biliary dilatation
        - C2: evidence of the aetiology on imaging (stricture, stone, stent etc.)
    - suspected diagnosis = 1 item in A plus 1 item in either B or C
    - definite diagnosis = 1 item in A, 1 item in B and 1 item in C
- investigations
    - bloods
        - FBC, U&E, LFT, CRP
        - blood cultures
        - amylase – often indicates involvement of the lower part of the common bile duct if raised
        - if available, bile fluid for culture
    - imaging
        - x-ray KUB and US abdomen may be done
        - contrast CT is most useful
        - MRI may be considered
- management
    - fluid resuscitation with correction of electrolyte abnormalities
    - antibiotics once blood cultures taken
        - effective against anaerobes and Gram negative bacteria
    - correct coagulation
    - emergency biliary drainage if there is:
        - shock
        - disturbance of consciousness
        - acute lung injury
        - acute kidney injury
        - hepatic injury
        - DIC
- management according to severity
    - stage I
        - antimicrobial therapy
        - percutaneous, endoscopic or operative intervention for non-responders depending on aetiology
    - stage II
        - early percutaneous or endoscopic drainage with a T-tube
        - more definitive surgery depending on aetiology once the patient is stabilised
    - stage III
        - treatment of organ failure with ventilatory support, vasopressors etc.
        - urgent percutaneous or endoscopic drainage or, in severe cases, decompression of the bile duct with a T-tube
        - definitive treatment once clinical picture improves

- prognosis
    - mortality between 17% and 40% depending on co-morbidities and age
    - early endoscopic drainage after stabilisation significantly improves prognosis

SuC7 cholecystitis

- overview
    - inflammation of the gallbladder
    - causes – mucosal injury
        - mechanical
        - chemical
        - infectious
    - complications
        - pancreatitis
        - ascending cholangitis
        - gallbladder empyema
        - gangrene
    - causative bacteria
        - Enterobacteriaceae (E coli, Klebsiella)
        - enterococci
        - bacteroides
        - clostridium
        - strep
        - staph
- clinical features
    - symptoms
        - RUQ and epigastric pain
        - radiation to left upper back
        - persists beyond 6 hours
        - often occurs between 21:00 and 04:00
        - nausea and vomiting
    - signs
        - dull, poorly localised pain, becoming sharp, well-localised mid-upper abdominal pain
- risk factors
    - increased age
    - female
    - parity
    - obesity
    - diabetes
    - profound weight loss
    - fasting
    - cystic fibrosis
    - malabsorption syndromes
    - familial
    - medications (e.g. contraceptive pill, clofibrate)
- investigations
    - bedside

        - ECG to rule out MI
    - laboratory
        - WCC, bilirubin and LFTs – often normal
        - chronic anaemia (haemolysis)
        - amylase – rule out pancreatitis
        - beta hCG in women of child-bearing age
    - imaging
        - AXR
            - 15% of stones visible
        - CXR
            - rule out right lower lobe pneumonia
        - US
            - 94% sensitivity and 78% specificity
            - better than CT
- management
    - IV fluids
    - nil by mouth
    - opioid analgesia
    - antiemetic
    - antibiotics
    - cholecystectomy

SuC8 diverticular disease

- background
    - diverticula most common in the sigmoid and descending colon but can be anywhere in the GI tract
    - it is a herniation of mucosa through the thickened colonic muscle
    - vary from single diverticulum to hundreds
    - typically 5-10mm in diameter, may exceed 2cm
    - definitions:
        - diverticulosis is the presence of asymptomatic diverticula
        - diverticular disease is diverticula with symptoms
        - diverticulitis is evidence of diverticular inflammation (fever, tachycardia) with or without localised symptoms and signs
- epidemiology
    - occurs in 5-10% of people 45 and older; occurs in 80% of people 85 and older
    - 75% have asymptomatic diverticulosis
    - rare before age 40
    - rare in rural Africa and Asia
- risk factors
    - age over 50
    - low dietary fibre
    - obesity
    - complicated diverticular disease more frequent in:
        - smokers
        - patients who use NSAIDs and paracetamol
        - obese

        - low fibre diet
- presentation
    - uncomplicated diverticular disease
        - frequently an incidental finding
        - can present with nonspecific abdominal complaints
            - lower abdominal pain, usually left sided
        - pain exacerbated by eating and diminished with defecation or passing flatus
        - may have other symptoms such as bloating, constipation, rectal bleeding
        - examination may reveal fullness or mild tenderness in the left lower quadrant
    - diverticulitis
        - presents with left lower quadrant pain
            - Asian patients usually present with right lower quadrant pain as diverticula are predominantly right sided
        - pain may be intermittent or constant and may be associated with change in bowel habits
        - most patients have fever and tachycardia
            - hypotension and shock are unusual
        - may have anorexia, nausea and vomiting
        - on examination, localised tenderness and sometimes a palpable mass
        - bowel sounds often reduced
        - rectal examination may reveal tenderness or a mass, especially with a low lying pelvic abscess
        - 5% of patients with diverticulitis develop complications:
            - perforation, abscess, fistula, stricture, obstruction
            - abscess
                - may be pericolic or more extensive
                - clinical signs include tender mass or persistent fever despite an adequate trial of antibiotics
                - may have features of sepsis
            - perforation
                - free perforation into the peritoneum causes frank peritonitis
                - rare, but can be life-threatening
            - obstruction
                - partial colonic obstruction or pseudo-obstruction may occur
            - stricture
                - recurrent episodes may cause progressive fibrosis and stricturing of the colonic wall, eventually leading to complete obstruction
            - fistula
                - colovesicular fistulas often present with pneumaturia and faecaluria
                - passage of stool or flatus via the vagina is pathognomic of colovaginal fistula
                    - may also present with frequent vaginal infections or copious vaginal discharge
- haemorrhage

    - diverticular bleeding is a common cause of lower GI haemorrhage
    - can be severe in 3-5% of patients
    - site may be in the proximal colon
    - presentation
        - usually abrupt painless bleeding
        - may have mild lower abdominal cramps or the urge to defecate followed by passage of a large amount of red or maroon blood or clots
        - melaena may occur but is uncommon
        - haemorrhage ceases spontaneously in 70-80%
            - rebleeding rates range from 22-38%
- investigations
    - asymptomatic diverticulosis
        - no investigations required
    - diverticular disease
        - if symptomatic, may require thorough investigation, including colonoscopy to confirm diagnosis and rule out other causes, particularly bowel cancer
        - FBC should be normal in uncomplicated disease
            - WCC is often raised in diverticulitis or abscess
            - bleeding may cause raised platelets and anaemia
    - suspected complicated diverticulitis
        - FBC, U&E, CRP
        - contrast CT within 24 hours of admission if inflammatory markers raised
            - non-contrast CT, MRI or US are alternatives if not possible
        - if inflammatory markers not raised, consider other diagnoses
    - fistulas
        - cystoscopy, cystography, contrast radiographs or methylene blue studies can show colovesicular fistula tracts
    - haemorrhage
        - flexible sigmoidoscopy initially to rule out obvious rectosigmoid lesion
        - if no cause identified, further assessment with non-invasive (nuclear scintigraphy) or invasive (angiography, colonoscopy) techniques can be undertaken to localise and treat the bleeding source
- management
    - asymptomatic disease
        - no treatment or follow up
        - high fibre diet may have prophylactic benefit, with adequate fluid intake
            - gradual increase in fibre may reduce flatulence and bloating
        - risk of perforation may be increased by NSAID and long term opiate use
    - diverticular disease
        - admission for significant blood loss from rectal haemorrhage
            - may require transfusion
        - high fibre diet of whole grains, fruit and vegetables
            - may take several weeks for the benefits to show
            - should be maintained for life if tolerated
        - adequate fluid intake

        - bulk forming laxatives (e.g. ispaghula, methylcellulose) may be beneficial if high fibre diet not effective or acceptable or if constipation or diarrhoea occurs
        - paracetamol for pain
        - an antispasmodic may help abdominal cramping
        - consider alternative diagnoses in patients not responding to management
    - diverticulitis
        - admission criteria:
            - pain not manageable with paracetamol
            - hydration not maintained with oral fluids
            - oral antibiotics not tolerated
            - frailty or significant comorbidity
            - rectal bleeding that may require transfusion
            - symptoms persisting after 48 hours of conservative management at home
            - features of complicated diverticulitis occur
        - for people managed at home:
            - consider a no antibiotic strategy with advice to seek further help if symptoms persist or worsen
            - oral antibiotics for patients who are systemically unwell, immunosuppressed or have significant comorbidity
                - one week of co-amoxiclav as first line
            - paracetamol for pain
            - clear liquids only initially, with solid foods reintroduced gradually as symptoms improve over 2-3 days
    - for people managed in hospital:
        - IV antibiotics for suspected complicated diverticulitis
            - co-amoxiclav first line
        - review in 48 hours or after scanning and switch to oral antibiotics when possible
    - abscess formation
        - pericolic abscesses <3cm can generally be treated conservatively with antibiotics and bowel rest
        - CT guided percutaneous drainage may be considered
        - if surgery required, primary anastomosis with or without diverting stoma or Hartmann's procedure are considered
    - fistulas
        - colovesical fistulas – single-stage resection with fistula closure suitable for most patients
        - colovaginal fistula – surgical resection of the diseased colon with repair of the vagina
    - obstruction
        - small bowel obstruction or ileus usually improves as inflammation subsides with effective treatment
        - strictures in which malignant disease cannot be excluded should be resected
            - trial of endoplasmic balloon dilation can be attempted if neoplasm can be excluded

        - stenting can provide temporary decompression allowing for subsequent resection
    - haemorrhage
        - may require fluid and blood products
        - colonoscopy should be performed once bleeding settles to establish source and exclude neoplasia
        - angiography with intra-arterial vasopressin can control haemorrhage in 90%, which is temporary but can allow time to prepare for surgery
        - surgery is usually reserved until endoscopic or angiographic treatments fail
            - segmental resection if bleeding site identified, otherwise subtotal colectomy may be required
        - surgical resection may be recommended after a second bleeding episode as risk of a third episode is as high as 50%
    - surgery
        - 15-30% of patients will need surgery
        - indications:
            - purulent or faecal peritonitis
            - uncontrolled sepsis
            - fistula
            - obstruction
            - inability to exclude carcinoma
        - free perforation with generalised peritonitis is uncommon but has a high mortality rate (up to 35%)
            - requires urgent surgical intervention
            - options include laparoscopic lavage and resectional surgery

SuC9 haemorrhoid disease

- haemorrhoids
    - enlarged vascular cushions in the anal canal
        - the anal canal contains three fibrovascular cushions
        - passing repeated hard stools and straining probably weakens the supporting tissues causing the cushions to prolapse
        - they can become distended and engorged with blood
    - classification
        - internal – above the dentate line
        - external – below the dentate line
    - classification in terms of degree of prolapse
        - 1st degree – completely internal
        - 2nd degree – prolapse but reduce spontaneously
        - 3rd degree – prolapse but reduce manually
        - 4th degree – cannot be reduced once prolapsed
    - normally painless unless thrombosed
    - tend to cause bright red blood on stool or in pan
    - management
        - most can be discharged to GP care or OP surgical follow up
        - fibre supplements to improve symptoms and prevent straining
        - good anal hygiene

        - little evidence to support topical local anaesthetic or steroid creams
        - urgent referral or admission for:
            - profuse haemorrhoidal bleeding
            - thrombosed haemorrhoids
                - although acutely painful most can be managed conservatively at home with analgesia, ice, bed rest, stool softeners
                - symptoms usually settle within 2 weeks
            - necrotic or gangrenous external haemorrhoid
                - may need surgical excision
            - patients with 4th degree haemorrhoids
            - suspected malignancy
        - surgical management
            - rubber band ligation as outpatient
                - effective and relieves symptoms in 80% of 1st, 2nd and 3rd degree haemorrhoids
            - haemorrhoidectomy
                - for 4th degree or failure of conservative therapy
                - traditional excision haemorrhoidectomy is more effective than newer staple techniques in terms of recurrence

SuC10 hernias

- background
    - protrusion of abdominal contents through the fascia of the abdominal wall
    - always contain a portion of peritoneal sac
        - may contain viscera, usually small bowel and omentum
    - male: female ratio of groin hernias is 8:1
- risk factors
    - infants
        - prematurity
        - male sex
    - adults
        - male sex
        - obesity
        - constipation
        - chronic cough
        - heavy lifting
- presentation
    - swelling in the groin
        - may appear with lifting and be accompanied by sudden pain
    - indirect hernias are more prone to cause pain in the scrotum and a dragging sensation
    - cough impulse (increase in swelling)
    - hernia may not be visible if reduced
    - if a lump is present it may be reducible
        - with time, hernias enlarge and become more difficult to reduce due to the formation of fibrous adhesions

        - when it can no longer be reduced, it is irreducible or incarcerated
            - can occur at any time, as can strangulation, when visceral contents of the hernia become twisted or entrapped by the narrow opening
                - this compromises the blood supply, causing swelling and eventually infarction
                - strangulation usually leads to bowel obstruction
- classification
    - indirect inguinal hernia
        - bowel passes through the inguinal canal via a congenital weakness of the internal inguinal ring
        - runs laterally to the inferior epigastric vessels
        - most common type
    - direct inguinal hernia
        - hernia exits the abdominal cavity directly through the deep layers of the abdominal wall
        - runs medially to the inferior epigastric vessels
        - uncommon (more common in the elderly)
    - femoral hernia
        - abdominal contents pass through the femoral canal just below the inguinal ligament
        - rare
        - almost exclusively in women because of the wider bone structure of the pelvis
            - incidence highest in middle aged and elderly women, especially if parous
        - high risk of strangulation (22% at 3 months, 45% at 21 months) so should be electively repaired at soon as possible
        - may not be noticed by the patient prior to strangulation, particularly in obese patients
- assessment
    - examine both standing and lying and ask the patient to cough or strain
    - insert a finger through the top of the scrotum into the external inguinal ring and palpate for a lump when coughing
- investigation
    - ultrasound
    - CT or MRI may be used
- management
    - if hernia is small, reassurance only
        - with the consideration that they may worsen at any time
    - episodes of pain and tenderness suggest the need for urgent intervention
    - emergency surgery required if the episode is prolonged or severe
        - involves reduction or excision of the sac and closure of the defect with minimal tension
            - mesh repair is often used and may be laparoscopic or open
- complications
    - recurrence (1%, mostly within 5 years of operation); increased risk in:
        - children under 1 year

        - elderly
        - after incarcerations
        - ongoing increased intra-abdominal pressure
        - growth failure
        - prematurity
        - chronic respiratory problems
        - girls with sliding hernias
    - infarcted testis or ovary with atrophy
    - wound infection
    - bladder injury
    - intestinal injury
    - hydrocoele from fluid accumulation in the distal sac – usually resolves spontaneously, sometimes requires aspiration

SuC11 intussusception

- adults
    - background
        - accounts for 1-5% of cases of intestinal obstruction
        - part of the GI tract invaginates or telescopes into a neighbouring portion
        - there is usually a lead point, which is the cause of it
    - types of case
        - ileo-ileal
            - affecting only the small bowel
            - more common than ileo-colic
        - colo-colic
            - affecting only the large bowel
        - ileo-colic
            - small bowel is pushed into the large bowel
        - ileo-caecal
            - small bowel is pushed into the caecum
    - causes
        - malignancy (54-69%)
            - primary neoplasm (e.g. bowel carcinoma, lymphoma, polyps, lipomas)
            - metastatic deposits (rare, e.g. renal cell carcinoma)
        - Meckel's diverticulum
        - abnormal peristalsis (secondary to ulceration)
        - heterotopic pancreatic tissue
        - endometriosis
        - inflammatory bowel disease
        - adhesions
        - association with enterovirus infection
        - association with DKA (possibly by altering digestive tract motility)
    - risk factors
        - cystic fibrosis
        - Roux-en-Y oesophagojejunostomy (e.g. for obesity)
        - Meckel's diverticulum

        - Peutz-Jegher's syndrome
        - familial polyposis coli
    - presentation
        - typically recurrent non-specific abdominal pain
        - nausea and vomiting in 20%
        - change in bowel habit
    - examination
        - abdominal distension
        - palpable mass
        - decreased or absent bowel sounds
        - can present as acute abdomen
        - bowel obstruction is uncommon
    - investigations
        - x-ray
            - not usually helpful
            - may show a soft tissue mass +/- bowel obstruction
        - barium enema
            - useful in colonic or ileo-colic intussusception with 'cup-shaped' filling defect
        - ultrasound
            - may show 'doughnut' or 'bull's eye' when the intussusception is seen transversely or 'pseudo kidney' or 'hayfork' sign in longitudinal section
        - CT
            - most effective and accurate diagnostic technique
            - may show a 'target lesion' in the distal ileum or ascending colon
        - colonoscopy
            - may visualise the intussusception and can be used to reduce it
                - depends on site of problem
            - may be better at detecting neoplastic mass as the lead point
            - biopsy is not recommended due to risk of perforation
    - management
        - observation
            - especially in conditions that alter motility
        - operative
            - reduction and resection
            - reduction before resection may lead to seeding of malignant cells, risk of perforation and risk of complications at the anastomosis site
                - can be used for benign lesions or to limit the extent of resection
            - large bowel intussusceptions are more likely to be malignancy (60% risk), so should probably be resected
    - complications
        - GI haemorrhage
            - from ileal ulcerations or mechanical trauma due to repeated intussusception
        - bowel obstruction +/- perforation

        - sepsis
        - shock (septicaemic or haemorrhagic)
- children
    - epidemiology
        - male:female ratio 3:2
        - two thirds of patients are under 1
            - peak age is 5-10 months
        - most common cause of intestinal obstruction in patients aged 5 months to 3 years
        - accounts for up to 25% of abdominal emergencies in children up to age 5
    - presentation
        - usually sudden onset
            - may be more insidious in older children
        - paroxysms of abdominal pain (about every 10-20 minutes) +/- crying
            - child may initially appear well between paroxysms
        - early vomiting, rapidly becoming bile stained
        - neurological symptoms can occur
            - lethargy, hypotonia, sudden alterations of consciousness
        - may be a palpable 'sausage shaped' mass
            - often in the right upper quadrant
        - may be absence of bowel in the right lower quadrant (Dance's sign)
        - dehydration, pallor, shock
        - irritability, sweating
        - later, mucoid and bloody 'redcurrant' stools
        - late pyrexia
    - causes and associated conditions
        - non-pathological lead point (>90%)
            - viral (50%)
                - rotavirus, adenovirus, HHV6
                - amoebomata, shigella, yersinia
                - Peyer's patch hypertrophy
        - pathological lead point (<10%)
            - Meckel's diverticulum
            - polyps and Peutz-Jegher's syndrome
            - Henoch-Schönlein purpura (3%)
            - lymphoma and other tumours (3%)
            - reduplication (bowel wall is duplicated)
            - cystic fibrosis
            - inflamed appendix
            - ascariasis
            - nephrotic syndrome
            - foreign body
            - postoperative
                - rarely can occur after operation for intussusception
            - hyperperistalsis
            - exclusive breastfeeding
            - weight above average

        - rotavirus vaccine
        - abdominal tuberculosis
- investigations
    - FBC
        - may show neutrophilia
    - U&Es
        - may show dehydration
    - abdominal x-ray
        - may show dilated gas-filled proximal bowel, paucity of gas distally, multiple fluid levels
        - may be normal in the early stages
    - ultrasound
        - may show doughnut or target sign, pseudokidney/sandwich appearance
        - effective and may be considered the modality of choice
    - bowel enema
        - barium or air and water-soluble double contrast
    - CT/MRI
        - used more often in adults than children
- management
    - 'drip and suck' – NG tube and IV fludis
    - radiological
        - reduction (three tries for three minutes each) if no sign of peritonitis, perforation or shock
        - air enema or barium enema
    - laparotomy
        - indications:
            - peritonitis
            - perforation
            - prolonged history (>24h)
            - high likelihood of pathological lead point
            - failed enema
- complications
    - missed diagnosis
    - ischaemia of the intussusceptum
    - sepsis
    - necrosis
    - haemorrhage
    - perforation
    - peritonitis
    - failure of enema reduction
    - chronic intussusception – rare cause of failure to thrive
- prognosis
    - excellent with treatment
    - spontaneous resolution may also occur
    - post reduction recurrence rate 5-15%
    - mortality is 1% with treatment

SuC12 ischaemic bowel

- background
    - inadequate blood flow through the mesenteric circulation causes ischaemia then gangrene
    - mainly seen in people over 50 but may be seen in younger people with risk factors
- causes
    - arterial disease
        - embolism
            - MI
            - mitral stenosis
            - AF
            - endocarditis
            - mycotic aneurysm
            - prosthetic grafts
            - myxoma
        - thrombosis
            - plaque rupture
        - occlusive disease
            - atherosclerosis
            - vasculitis
            - autoimmune disease
        - non-occlusive disease
            - decreased perfusion
                - septic shock
                - hypovolaemia
                - vasopressors
                - pancreatitis
    - venous disease
        - intra-abdominal infection with portal pyaemia
        - hypercoagulable states
        - portal hypertension/mass effect from tumours
            - stasis
        - direct trauma from surgery
        - band adhesions
- history
    - abdominal pain
    - nausea and vomiting
    - diarrhoea
    - arterial embolic:
        - abrupt, painful
        - vomiting
        - diarrhoea
        - recent AF, MI, valvular heart disease
    - arterial occlusive:
        - associated with eating
        - pain out of proportion to physical findings
        - unresponsive to opioids

        - nausea and vomiting
        - distension
        - GI haemorrhage
    - arterial thrombosis:
        - often have history of mesenteric angina
        - associated with CCF, MI
    - non-occlusive disease:
        - associated with multi-organ dysfunction
    - venous thrombosis:
        - symptoms may have been present for weeks
        - may have history of hypercoagulability
- examination
    - tenderness
    - peritonism
    - palpable mass
    - bowel sounds absent or hyperactive
    - septicaemia
    - AF
    - heart murmurs
- investigations
    - bloods
        - unreliable for diagnosis
    - ABG
        - metabolic acidosis
        - elevated lactate
    - x-ray
        - exclude perforation
        - thumb printing
        - portal vein gas
    - CT
        - angiogram (96% sensitive, 94% specific)
        - pneumotosis intestinalis
        - portal vein gas
        - bowel or mesenteric oedema
        - abnormal gas patterns
        - streaking of mesentery
        - solid organ infarction
    - MRI/MRA
        - highly specific and sensitive
- management
    - IV fluids
    - nil by mouth
    - opioid analgesia
    - antiemetics
    - antibiotics
    - surgery
    - angiographical infused thrombolytics
    - angioplasty

    - heparin for venous thrombosis
- prognosis
    - mortality rate 50-90% with treatment, 90% if diagnosis missed
    - significant morbidity for survivors
- chronic mesenteric ischaemia
    - chronic atherosclerotic disease of the vessels supplying the intestines
        - also called intestinal angina
    - usually involves all three mesenteric arteries
    - low incidence
    - most common in females between 50 and 70 with risk factors for atherosclerosis
    - presentation is moderate-to-severe colicky or constant poorly localised pain
        - weight loss
        - postprandial pain
        - fear of eating
        - may have nausea, vomiting, bowel irregularity
    - may have vague tenderness disproportionate to severity of pain
    - arteriography is the gold standard investigation to show the site of arterial blockage or stenosis
        - mesenteric duplex ultrasonography may be considered
    - management:
        - conservative for asymptomatic patients
            - smoking cessation
            - antiplatelet therapy
        - symptomatic ischaemia is an indication for open or endovascular revascularisation
            - nutritional optimisation is important prior to surgery
    - most patients have poor quality of life with significant weight loss due to fear of abdominal pain on eating
- ischaemic colitis
    - caused by a compromise of the blood supply to the colon
    - causes:
        - thrombosis
            - inferior mesenteric artery thrombosis
        - emboli
            - mesenteric arterial emboli
            - cholesterol emboli
        - decreased cardiac output or arrhythmias
        - shock
        - trauma
        - strangulated hernia or volvulus
        - drugs:
            - digitalis
            - oestrogens
            - antihypertensive drugs
            - cocaine and methamphetamine
            - vasopressin
            - phenylephrine

                - pseudoephedrine
                - immunosuppressive agents
                - psychotropic agents
            - surgery:
                - cardiac bypass
                - aortic dissection and repair
                - aortoiliac reconstruction
                - colectomy with inferior mesenteric artery ligation
                - gynaecological operations
            - vasculitis
            - disorders of coagulation
            - long distance running
            - colonoscopy or barium enema
            - idiopathic
    - presentation
        - may be difficult to diagnose
            - non-specific symptoms such as acute abdominal pain
                - most likely LIF
            - nausea and vomiting
            - loose stool containing dark blood in the later stages
        - symptoms tend to occur within hours
    - investigations
        - metabolic acidosis
        - colonoscopy
            - blue, swollen mucosa sparing the rectum
        - abdominal x-ray
            - may show abnormal segment outlined with gas
        - barium enema
            - thumb printing in the early phase (may last for several days)
        - CT, MRI and angiography may also be used
    - management
        - medical
            - management of the cause of hypoperfusion – ischaemia may be transient
            - bowel rest
            - supportive care
            - broad spectrum antibiotics
        - surgical
            - repeat colonoscopy or CT angiography if symptoms not improving in 24-48 hours
            - urgent laparotomy for increasing abdominal tenderness with guarding, fever, uncontrolled bleeding, paralytic ileus – suggests infarction of the colon
    - prognosis
        - depends on location and extent of disease
        - severity and overall mortality higher in right sided disease
        - mortality around 22%

SuC13 lower gastrointestinal and rectal bleeding

- background
    - accounts for around 20% of GI haemorrhage cases
    - usually causes less haemodynamic instability than upper GI haemorrhage
    - when catastrophic, mortality rates are up to 21%
    - bleeding usually stops during initial resuscitation
- definition
    - bleeding distal to the ligament of Treitz
        - suspensory muscle of the duodenum that connects to the diaphragm
        - includes some of the small bowel, the colon and rectum
- history
    - may present with passage of bright red blood per rectum
    - may find it difficult to describe
        - clarify nature of blood in terms of frequency, colour, presence/absence of clots
    - fatigue/chest pain/palpitations/shortness of breath
    - abdominal pain
    - weight loss
    - fever
    - diarrhoea
    - vomiting
    - medication use
        - particularly anticoagulants/antiplatelets/NSAIDs
- examination
    - assessment of shock
        - dyspnoea
        - tachypnoea
        - tachycardia
        - postural drop in blood pressure
    - abdominal tenderness
        - may suggest inflammatory cause such as ischaemic colitis
    - PR examination
        - assess stool colour
        - presence of blood
        - anorectal lesions
- risk assessment
    - no well validated scoring system
    - BLEED criteria (high risk)
        - ongoing bleeding
        - hypotension (systolic <100mmHg)
        - abnormal clotting (PT >1.2s)
        - altered mental status
        - significant co-morbidities
    - Scottish National Guidelines committee criteria for discharge/admission
        - consider for discharge or OP follow up:
            - age <60 and
            - no evidence of haemodynamic compromise

                - no evidence of gross rectal bleeding
                - an obvious anorectal source od bleeding on rectal examination/sigmoidoscopy
            - consider admission:
                - age ≥60 or
                - haemodynamic disturbance or
                - evidence of gross rectal bleeding or
                - taking aspirin or an NSAID or
                - significant co-morbidity

- causes
    - diverticular disease
        - commonest cause of lower GI bleeding
        - uncommon under 40
    - inflammatory bowel disease
        - includes:
            - Crohn's disease
            - ulcerative colitis
            - ischaemic colitis
                - usually transient and reversible
            - infective colitis
                - requires stool culture to test for Salmonella, Shigella, Campylobacter
    - neoplasia
        - carcinoma and polyps can cause lower GI haemorrhage
            - more commonly presents with occult blood loss
    - benign anorectal disease
        - rarely life-threatening but can cause considerable distress
        - includes anal fissure and haemorrhoids
        - usually fresh blood on paper or surface of stool
    - angiodysplasia
        - acquired malformation of the intestinal blood vessels with ectatic vessels in the mucosa and submucosa
        - colonoscopy shows dilated vessels or cherry red, flat lesions
        - 80% occur in the right side of the colon
            - they may be multiple
            - can be associated with valvular heart disease and other systemic diseases
        - most commonly presents with iron deficiency anaemia and occult blood loss
            - due to slow but repeated bleeding episodes
        - incidental finding on colonoscopy in 5% of cases
        - overt bleeding is typically brisk, painless and intermittent
    - other
        - these include:
            - radiation injury
            - Meckel's diverticulum
            - other small bowel pathology
            - solitary rectal ulcers

                - portal colopathy
                - prostate biopsy sites
                - Dieulafoy lesions
                - endometriosis
                - colonic varices
        - 10-15% of patients with an apparent lower GI bleed in fact have an upper GI source
- investigation
    - bloods
        - U&Es
        - FBC
            - Hb and platelets
        - coagulation screen
        - LFTs
        - G&S or crossmatch depending on urgency
        - VBG
    - ECG
    - PR
        - +/- FOB
    - CXR
        - AXR is unlikely to be helpful unless to exclude pathologies such as toxic megacolon
    - nasogastric aspirate may be considered to exclude an upper GI source if there is suspicion of this
- management
    - initial resuscitation in the ED
    - blood and blood products as required
    - definitive treatment
        - often carried out at the same time as investigation
        - options include:
            - colonoscopy and treatment
                - low morbidity/mortality
                - yield of 72-86%
                - American Society of Gastroenterology recommend colonoscopy as first option for most lower GI bleeds
                - if high risk features or ongoing bleeding, it should be performed within 24 hours
                - can be difficult to visualise the mucosa during or directly after a bleed, making finding the bleeding point more challenging
                - coagulation (thermal or adrenaline) can be performed during the procedure, particularly for AVMs
                    - risk of perforation must be considered
                    - other options include haemoclips and band ligation
            - angiography and treatment
                - thought to be preferable in those with massive haemorrhage

        - success rates in identifying the source of the bleed vary from 40% to 85%
        - superselective embolization aims to decrease arterial blood flow, which reduces pressure to the bleeding site allowing haemostasis
        - NCEPOD recommends that hospitals which do not provide onsite IR should have a formalised regional network
    - other investigative options:
        - proctoscopy
            - should be done in all patients with rectal bleeding according to SIGN guidelines
        - technetium labelled red blood cell scanning
        - upper GI endoscopy
        - CT angiography
        - recto-sigmoidoscopy
    - medical therapy
        - no medical therapies
        - risk-benefit discussion for anticoagulants
        - low dose aspirin is usually continued and NSAIDs stopped
    - surgical therapy
        - required for a small minority (around 5%)
        - considered in massive bleeds or if other treatment options have failed
        - pre-operative location of the bleeding site is preferable to allow segmental resection
            - if not localised before or during the procedure a subtotal colectomy may be required

SuC14 pancreatitis

- definition
    - the pancreas is the largest gland in the body (12.5-15cm long)
    - endocrine and exocrine gland
        - endocrine
            - insulin
            - glucagon
            - somatostatin
        - exocrine
            - pancreatic juice containing digestive enzymes to help break down carbohydrate, proteins and fat in chyme
    - pancreatitis can have both localised and systemic effects
- phases of pancreatitis
    - phase 1
        - obstruction of bile or pancreatic ducts or direct toxicity
        - leads to premature activation of trypsin within the pancreatic acinar cells
        - activates a variety of pancreatic digestive enzymes
    - phase 2
        - intrapancreatic inflammation

        - can lead to necrosis, haemorrhage, infection or collection of fluid and formation of a pseudocyst
    - phase 3
        - extrapancreatic inflammation
    - in 10-20% of cases, phases 2 and 3 can lead to shock and SIRS
        - lung
            - hypoxia, ARDS, pleural effusions (normally left-sided)
        - kidney
            - acute renal failure and acute tubular necrosis
        - blood
            - coagulopathy, DIC, hypocalcaemia, hyperglycaemia
        - general
            - hypovolaemia and shock
- history
    - abdominal pain
        - typically epigastric, radiating to the back
        - typically sudden, severe and reaches a maximum level quickly
        - relieved by nothing and exacerbated by movement
        - nausea, retching, vomiting
    - 50% have had similar episodes in the past
    - risk factors
    - co-morbidities
    - drugs and alcohol
- examination
    - tender upper abdomen
        - may have guarding but usually not as intense as the level of pain would suggest due to the retroperitoneal position of the pancreas
    - jaundice
        - if there is also obstruction of the common bile duct
    - paralytic ileus
        - after first 12-24 hours
        - mild abdominal distension and absent bowel sounds
    - Cullen's and Grey-Turner's signs
        - Cullen's sign is bruising around the umbilicus
        - Grey-Turner's sign is flank bruising
        - rare and late signs of excessive pancreatic destruction
        - due to haemorrhage from the pancreas leading to blood in the abdominal cavity
    - signs of tetany
        - fasciculations
        - twitching
        - positive Trousseau's or Chvostek's test
        - hypocalcaemia can develop secondary to intra-abdominal fat necrosis
- risk assessment
    - Ranson criteria (mortality prediction)
        - at admission
            - age >55

                - WCC >16
                - glucose >11
                - AST >250
                - LDH >350
            - 48 hours into admission
                - calcium <2mmol/L
                - haematocrit fall >10%
                - arterial pO2 <8kPa
                - fluid sequestration >6 litres
                - base deficit >4mEq/L
                - urea rise >1.8mmol/L
    - Glasgow score
        - PaO2 <7.9kPa
        - age >55
        - WCC >15
        - calcium <2mmol/L
        - urea >16mmol/L
        - LDH >600
        - albumin <32g/L
        - glucose >10mmol/L
    - severity assessment
        - initial assessment
            - clinical impression of severity
            - BMI >30
            - pleural effusion on CXR
            - APACHE II score >8
        - 24 hours after admission
            - clinical impression of severity
            - APACHE II score >8
            - Glasgow score 3 or more
            - persisting organ failure, especially if multiple
            - CRP >150
        - 48 hours after admission
            - clinical impression of severity
            - Glasgow score 3 or more
            - CRP >150
            - persisting organ failure for 48 hours
            - multiple or progressive organ failure
- mortality rates
    - overall mortality rate is 5%
        - 3% in interstitial pancreatitis
        - 17% in necrotising pancreatitis
- differential diagnosis
    - perforated gastric or duodenal ulcer
    - biliary colic
    - mesenteric ischaemia or infarction
    - dissecting aortic aneurysm

    - intestinal obstruction
    - inferior wall myocardial infarction
- aetiology (GETSMASHED)
        - **g**allstones
        - **e**thanol
        - **t**rauma
        - **s**teroids
        - **m**umps
        - **a**utoimmune
        - **s**corpion venom
        - **h**ypothermia/hyperlipidaemia
        - **E**RCP
        - **d**rugs (azathioprine, thiazides, sodium valproate, tetracyclines)
    - 75-80% of cases are caused by gallstones or alcohol
- investigation
    - bloods
        - FBC, U&E, CRP, LFT, amylase, VBG, glucose, calcium
            - lipase is slightly more accurate than amylase if available
            - enzymes peak at 3-4 days so normal amylase does not rule out pancreatitis
        - ABG
        - urinalysis
            - plus beta hCG when indicated
        - ECG
        - CXR
- management
    - all patients should be treated aggressively until severity established
    - resuscitation
        - may require invasive monitoring and inotropes
    - opioid analgesia and antiemetic
    - nil by mouth
        - NG tube if evidence of ileus
    - urinary catheter with output monitoring
    - broad spectrum antibiotics if evidence of sepsis
    - admission under surgical team
        - with HDU/ICU input if needed
    - CT for patients with persisting organ failure, signs of sepsis or deterioration in clinical status after admission
    - early ERCP if gallstone obstruction identified as cause
    - surgery
        - rarely required
        - may be needed if pancreatitis complicated by pancreatic or peripancreatic necrosis
- chronic presentations
    - patients may present with recurrent acute pancreatitis
    - chronic pancreatitis is loss of pancreatic function secondary to inflammation
        - patients develop malabsorption, steatorrhoea and sometimes diabetes

- chronic pancreatitis can present to the ED as acute exacerbations or with chronic pain/weight loss/malabsorption
- management is usually analgesia, oral pancreatic enzyme supplements and blood glucose control
  - large amounts of regular opiates and insulin may be needed
- surgery to remove all or part of the pancreas and/or drainage procedures can sometimes be performed

SuC15 viscus perforation

- background
  - may occur at any anatomical location from the upper oesophagus to the anorectal junction
  - can progress rapidly to septic shock and multi-organ dysfunction if not identified
- aetiology
  - most common causes are peptic ulcers and sigmoid diverticulum
  - inflammatory or ischaemic
    - chemical
      - peptic ulcer disease
        - gastric or duodenal
      - foreign body
        - e.g. battery, caustic soda
    - infection
      - diverticulitis
      - cholecystitis
      - Meckel's diverticulum
    - ischaemia
      - mesenteric ischaemia
      - obstructing lesions
        - e.g. cancer (can also invade through the bowel wall), bezoar, faeces
        - results in bowel distension and subsequent ischaemia and necrosis
    - colitis
      - toxic megacolon
        - e.g. Clostridium difficile or ulcerative colitis
  - traumatic
    - iatrogenic
      - recent surgery
      - endoscopy
      - NG tube insertion
    - penetrating or blunt trauma
      - shear forces from acceleration-deceleration
      - high forces over small area (e.g. handlebar)
    - direct rupture
      - Boerhaave syndrome
- history
  - main feature is pain

        - typically rapid onset and sharp in nature
        - in thoracic perforation:
            - chest or neck pain
            - pain radiating to the back
            - typically worse on inspiration
    - systemic symptoms
        - malaise
        - vomiting
        - lethargy
        - may be respiratory symptoms with oesophageal perforation
- examination
    - patients look unwell and may have features of sepsis
    - features of peritonism – may be generalised or localised
        - peritonism throughout the abdomen implies generalised contamination and the need for urgent surgery
    - oesophageal perforation may cause pleural effusion and/or palpable crepitus
- investigations
    - FBC, U&E, CRP, LFT, amylase, VBG, G&S
        - WCC, CRP and amylase may be raised
    - urinalysis +/- beta hCG
    - imaging
        - erect CXR
            - shows free air under the diaphragm
            - pneumomediastinum or widened mediastinum may be present if perforation is thoracic
            - 3 in 10 patients with perforation will have no signs on CXR
        - CT
            - gold standard imaging
            - confirms free air, suggests location and cause
        - AXR
            - limited role due to availability of CT
            - features include:
                - Rigler's sign
                    - both sides of the bowel wall can be seen
                        - free intra-abdominal air acts as additional contrast
                - psoas sign
                    - loss of the sharp delineation of the psoas muscle border secondary to fluid in the retroperitoneum
- management
    - resuscitation
    - broad spectrum antibiotics
    - nil by mouth
    - consider NG tube
    - analgesia
    - surgical intervention
        - identify and where possible manage underlying cause

        - management of perforation
            - repair of perforated peptic ulcer with an omental patch
            - resection of perforated diverticulae
            - thorough washout
    - conservative management
        - for select physiologically well patients including:
            - localised diverticular abscess/perforation with only localised peritonitis and tenderness and no evidence of generalised contamination on CT
            - patients with a sealed upper GI perforation on CT without generalised peritonism
            - elderly frail patients with extensive co-morbidities who would be unlikely to survive surgery
- complications
    - infection
        - peritonitis and sepsis
        - haemorrhage

SuC16 volvulus

- background
    - a twisting of a loop of intestine around its mesenteric attachment
        - results in a closed loop bowel obstruction
        - bowel can become ischaemic due to compromised blood supply, leading to necrosis and perforation
    - most occur at the sigmoid colon
        - can also occur at stomach, caecum, small intestine, transverse colon
    - the sigmoid colon has a long mesentery, which increases with age
        - makes the segment more prone to twisting
    - caecal volvulus is the second most common type
        - bimodal onset in the 10-29 and 60-79 age groups
            - the younger group may have intestinal malformation or excessive exercise as the underlying cause
            - management is always surgical with laparotomy and ileocaecal resection
- risk factors
    - increasing age
    - neuropsychiatric disorders
    - nursing home residents
    - chronic constipation or laxative use
    - male gender
    - previous abdominal operations
- clinical features
    - presents with clinical features of bowel obstruction
    - vomiting is usually a late sign
        - due to the sigmoid colon being located distally in the GI tract
    - early colicky pain, abdominal distension and absolute constipation
    - onset is rapid

    - degree of distension can be large
    - on examination
        - very tympanic abdomen
        - signs of perforation or peritonism indicates ischaemia or perforation
- differential
    - alternative causes of bowel obstruction
    - severe constipation
    - pseudo-obstruction
    - severe sigmoid diverticular disease
- investigations
    - bloods
        - FBC, U&E, LFT, CRP, amylase, G&S, calcium, TFTs
            - look for causes of pseudo-obstruction
    - CT
        - abdo-pelvis with contrast
        - identifies site and cause
        - demonstrates dilated colon with 'whirl sign'
    - AXR
        - shows 'coffee bean' sign arising from left iliac fossa in 60-75% of cases)
        - if ileo-caecal valve is incompetent there will also be signs of small bowel dilatation
- management
    - initial management as per any bowel obstruction
        - including resuscitation and analgesia
    - conservative
        - most patients are treated conservatively initially
            - decompression by sigmoidoscope and insertion of flatus tube
                - the patient is placed in the left lateral position and the scope guided into the rectum
                - it is manoeuvred to locate the twisted bowel and when in the correct position there is a rush of air and faeces
                - a flatus tube is left in situ for up to 24 hours to allow for continued passage of contents and aid recovery of the affected area
    - surgical management
        - indications:
            - colonic ischaemia or perforation
            - repeated failed attempts at decompression
            - necrotic bowel noted at endoscopy
        - usually laparotomy for a Hartmann's procedure
        - patients with recurrent volvulus may have an elective procedure
            - usually sigmoidectomy with primary anastomosis
- complications
    - bowel ischaemia and perforation
    - recurrence (in up to 90%)
    - surgical mortality is high as patients are usually old and frail with multiple co-morbidities

TRAUMA

TP1 head injury

- background
    - head injury can be from blunt or penetrating trauma
    - 70% of head injuries are in males and up to 50% in children under 15
    - falls and assaults are the most common cause of minor head injury, followed by road traffic accidents
    - alcohol may be involved in up to 65% of adult head injuries
    - indications for concurrent c-spine immobilisation include:
        - GCS <15 at any time since injury
        - neck pain or tenderness
        - focal neurological deficit
        - paraesthesia in the extremities
        - any other clinical suspicion of cervical spine injury
- indications for ED attendance
    - history of head injury
        - high-energy head injury
            - e.g. diving accident, high speed motor vehicle collision
        - GCS <15 at any time since injury
        - any loss of consciousness
        - any focal neurological deficit since the injury
        - amnesia for events before or after the injury
        - persistent headache since the injury
        - any vomiting episodes since the injury
        - any seizure since the injury
        - irritability or altered behaviour, especially in infants or young children
        - any suspicion of skull fracture or penetrating head injury
        - other visible trauma to the head causing concern
    - past medical history
        - age ≥65
        - any previous cranial neurosurgical interventions
        - history of bleeding or clotting disorder
        - current anticoagulation therapy such as warfarin or DOAC
    - other concerns
        - suspicion of non-accidental injury
        - current drug or alcohol intoxication
        - adverse social factors (e.g. nobody to supervise at home)
        - continuing concern by a healthcare professional about the diagnosis
        - continuing concern by the injured person or their carer about the diagnosis
- criteria for hospital admission
    - adult
        - new clinically significant abnormalities on imaging
        - not returned to GCS 15 after imaging, regardless of imaging results
        - patient fulfils criteria for CT scanning but this cannot be done
        - continuing worrying signs (e.g. persistent vomiting, severe headache)
        - other sources of concern

                - drug or alcohol intoxication
                - other injuries
                - shock
                - suspected non-accidental injury
                - meningism
                - CSF leak
        - children
            - history of loss of consciousness
            - neurological abnormality, persisting headache or vomiting
            - clinical or radiological evidence of skull fracture or penetrating injury
            - difficulty in making a full assessment
            - suspicion of non-accidental injury
            - other significant medical problems
            - not accompanied by a responsible adult or social circumstances considered unsatisfactory
- discharge advice
    - should be verbal and written
    - should include information on:
        - details of the injury
            - nature
            - severity
        - warning signs that warrant further medical investigation
            - increasing drowsiness
            - worsening headache
            - confusion or strange behaviour
            - two or more bouts of vomiting
            - focal neurological problem
                - e.g. limb weakness
            - dizziness, loss of balance or convulsions
            - any visual problem such as blurring of vision, or double vision
            - blood, or clear fluid, leaking from the nose or ear
            - unusual breathing patterns
        - responsible adult to stay with the patient during the first 24 hours following injury
        - how long recovery is likely to take and what this will involve
            - including when to go back to work and undertake everyday activities including sports
        - potential complications
        - who to contact if further help is needed
        - available support organisations
- investigations
    - CT scan
        - adults
            - within 1 hour
                - GCS <13 when first assessed
                - GCS <15 two hours after injury
                - suspected open or depressed skull fracture

        - signs of base of skull fracture
        - post-traumatic seizure
        - focal neurological deficit
        - >1 episode of vomiting
    - within 8 hours
        - coagulopathy or on oral anticoagulants
- children under 16
    - within 1 hour
        - clinical suspicion of non-accidental injury
        - post-traumatic seizure (no past medical history of epilepsy)
        - GCS <14 on initial assessment (or <15 if <1 year)
        - GCS <15 two hours after injury
        - suspected open or depressed skull fracture or tense fontanelle
        - signs of base of skull fracture
        - focal neurological deficit
        - aged <1:
            - bruise, swelling or laceration >5cm on head
    - within 1 hour if more than one of the following:
        - witnessed loss of consciousness >5 minutes
        - amnesia (anterograde or retrograde) >5 minutes
        - abnormal drowsiness
        - ≥3 discrete episodes of vomiting
        - dangerous mechanism or injury
            - high speed RTA
            - fall from >3 metres
            - high speed projectile
    - one of the above present – observe for 4 hours and scan within 1 hour if:
        - GCS <15
        - further vomiting
        - abnormal drowsiness

- indications for neurosurgical opinion
    - new surgically significant abnormalities on imaging
    - persisting coma (GCS ≤8) after initial resuscitation
    - unexplained confusion which persists for more than 4 hours
    - deterioration in GCS after admission
        - with greater attention to deterioration in motor response
    - progressive focal neurological signs
    - seizure without full recovery
    - depressed skull fracture
    - definite or suspected penetrating injury
    - CSF leak
- primary injury
    - contusions
        - bruises to brain parenchyma
    - haematomas

        - extradural haematoma
        - subdural haematoma
        - intraparenchymal
        - intraventricular
        - subarachnoid haemorrhage
    - diffuse axonal injury
    - direct cellular damage
        - neurons
        - axons
    - tearing and shearing of tissues
- secondary injury
    - brain oedema causes increased ICP
        - this compresses the tissue causing ischaemia with direct compression of the vasculature causing brain tissue herniation and brain death
    - expansion of the original injury
        - predominantly metabolic insult
            - calcium and sodium shifts
            - mitochondrial damage
            - production of free radicals
        - ultimately leads to damage to axonal integrity and axonal transport
            - enzyme activity leads to apoptosis
        - microscopic structural injury is often unidentifiable on CT or MRI
    - cerebral blood flow and autoregulation
        - vasoconstriction
        - no good way to measure cerebral blood flow
            - CPP used as a surrogate
                - amount of pressure needed to perfuse the brain
                - CPP = MAP-ICP
                    - when ICP increases, CPP decreases
                    - normal ICP is 15 in adults, <10 to 15 in children, 1.5-6.0 in infants
        - autoregulation allows the body to control the cerebral blood flow
            - autoregulatory mechanism is damaged in most traumatic brain injury patients
- GCS
    - eye opening
        - 4: spontaneously opens
        - 3: opens to command
        - 2: opens to pain
        - 1: does not open
    - verbal
        - 5: oriented
        - 4: confused speech
        - 3: inappropriate words
        - 2: incomprehensible sounds
        - 1: no response
    - motor

        - 6: obeys commands
        - 5: localises to pain
        - 4: withdraws from pain (normal flexion)
        - 3: decorticate posturing (abnormal flexion)
        - 2: decerebrate posturing (extension)
        - 1: no response
- classification of head injury
    - GCS 14-15: mild
    - GCS 9-13: moderate
    - GCS 3-8: severe
- Cushing's reflex
    - more common in children than adults
    - consists of:
        - hypertension
        - bradycardia
        - respiratory irregularity
- investigation
    - non-contrast CT +/- face/orbit CT, c-spine CT
    - blood glucose
        - hypoglycaemia increases risk of secondary brain injury and lowers seizure threshold
    - FBC
        - consider transfusion if Hb <70
        - consider transfusion for thrombocytopenia if evidence of intracranial haemorrhage
    - coagulation screen
    - electrolytes
        - seizure threshold may be reduced with hyponatraemia, hypomagnesaemia, hypophosphataemia
- management of severe head injury
    - main goals
        - prevent intracranial hypertension
        - prevent secondary brain insults
        - maintain cerebral perfusion pressure >70mmHg
        - optimise cerebral oxygenation and blood flow
        - monitor for increased ICP
        - monitor for herniation
        - maintain PaO2 >8kPa
        - prevent hyperthermia
        - prevent hypotension
        - correct coagulopathy
        - tranexamic acid
            - GCS ≤12 OR intracranial bleeding on CT scan AND ≤ 3 hours since time of injury
            - loading dose 1g over 10 minutes, then maintenance dose 1g over 8 hours
            - NNT 76.9

- airway
  - avoid hypoxia (SpO2 <90%, PaO2 <8kPa)
  - consider early endotracheal intubation
    - pre-oxygenation
      - hyperoxia can be harmful but targeting normoxaemia immediately after securing airway probably outweighs potential harm of hypoxia during intubation
    - pre-treatment
      - short acting opioids can be considered in haemodynamically stable patients
    - induction
      - choice of agents remains controversial
        - etomidate is well-studied with a stable haemodynamic profile
        - ketamine is a good choice in patients with haemodynamic compromise
- breathing
  - avoid hyperventilation
    - hypocapnia induces cerebral vasoconstriction
    - target low normal pCO2
- circulation
  - avoid hypotension (SBP <90mmHg)
  - fluid resuscitation with saline +/- vasopressors
  - no apparent benefit to initial administration of hypertonic saline
- management of impending herniation
  - hyperosmolar therapy indicated for patients with objective elevations of intracranial pressure or signs of impending or ongoing herniation
  - hypertonic saline and mannitol are both effective
- management of increased ICP
  - head of bed elevation
    - 30 degrees or reverse Trendelenburg will lower ICP
    - keep head and neck in neutral position, improving cerebral venous drainage
    - avoid compressing IVJ or EVJ with tight collars or fixation of ETT
  - maintain cerebral perfusion
    - no class 1 evidence for optimal CPP
    - aim for Hb >100
    - fluids and vasopressors if needed for CPP 70-80mmHg
      - mortality increases 20% for each 10mmHg loss of CPP
      - avoid dips <70mmHg – associated with cerebral ischaemia
      - vasopressors
        - phenylephrine increases CPP without increasing ICP in animal models
          - avoid if patient is already bradycardia
      - IV fluids
        - maintain euvolaemia
        - initial resuscitation with 0.9% sodium chloride

                - then consider hypertonic saline/mannitol
            - avoid Hartmann's as slightly hypotonic
            - gradual correction of severe hypernatraemia
    - osmotherapies
        - choice of agent should be coordinated with neurosurgery and take patient's blood pressure into account
            - mannitol may cause hypotension due to osmotic diuresis
        - mannitol
            - if SBP >90mmHg
            - bolus 20% at 0.25-1g/kg as rapid infusion over 15-20 minutes
            - reduces ICP within 30 minutes
            - duration of action 6-8h
            - monitor input/output to maintain euvolaemia during expected diuresis and use 0.9% sodium chloride to volume replace
            - do not use continuous infusions
                - mannitol crosses the blood brain barrier after prolonged administration and contributes to cerebral oedema
                - consider hypertonic saline for further boluses
        - hypertonic saline
            - obtain baseline serum osmolarity and sodium
            - 250ml bolus of 3% can be delivered through a peripheral line
            - target sodium 145-155mmol/dL
    - prevent cerebral vasoconstriction
        - hyperventilation does not improve mortality and should only be used as a temporising measure
            - should only be used if reduction in ICP necessary without any other means or ICP elevation refractory to all other treatments:
                - sedation
                - paralytics
                - CSF drainage
                - hypertonic saline, osmotic diuretics
            - should only be used at the lower end of normal for up to 30 minutes, no longer if it can be avoided
            - severe hypocarbia decreases cerebral blood flow to ischaemic levels
    - seizure control
        - treat immediately with benzodiazepines and anti-epileptic drugs
        - consider propofol for post-intubation sedation
        - seizure prophylaxis reduces seizures but does not improve long-term outcome
        - risk factors for post-traumatic seizures:
            - GCS <10 initially
            - cortical contusion
            - depressed skull fracture
            - subdural haematoma
            - extradural haematoma
            - subarachnoid or intracerebral haemorrhage
            - penetrating head injury

                - seizure within 24 hours of injury
            - any apparent and EEG confirmed seizures should be treated
                - prophylaxis should be considered for patients with the above risk factors
                - levetiracetam
                    - 20mg/kg IV followed by 1g BD IV for 7 days
                    - 40mg/kg(30-60mg/kg) up to 4.5g can be used
    - decrease metabolic rate
        - adequate sedation and analgesia
        - avoid hyperthermia and treat fever aggressively
    - other critical care measures
        - DVT prophylaxis with compression devices and avoidance of anticoagulation
        - consider stress ulcer prophylaxis with PPI
        - good glycaemic control (tight maintenance not supported)
        - steroids are contraindicated in severe traumatic brain injury
        - routine paralysis is not indicated but may be required for refractory ICP elevation
- consideration for neurosurgical intervention
    - intracranial pressure monitoring for salvageable patients with severe traumatic brain injury and abnormal CT (haematoma, contusion, swelling, herniation, compressed basal cisterns)
    - extradural haematoma
        - volume >30ml
        - GCS <8
        - aniscoria
    - subdural haematoma
        - >10mm width with >5mm midline shift
        - GCS <8
        - drop in GCS >2
    - posterior fossa haemorrhage
        - mass effect (distortion of 4th ventricle, obstructive hydrocephalus)
    - depressed skull fracture
        - depth >thickness of cranium
        - dural penetration
        - pneumocephalus

TP2 spinal injury

- primary spinal cord injury
    - results from blunt or penetrating mechanisms at the time of the initial traumatic event
    - fractures, dislocations, haematomas and soft tissue swelling directly injure the spinal cord via mass effect, disrupted blood supply or transection
- secondary spinal cord injury
    - occurs due to:
        - mechanical instability contributing to ongoing direct injury
        - insults from other factors such as hypoxia and hypoperfusion
        - focus of acute management

- classification of spinal cord injury
    - American Spinal Injury Association (ASIA) classification, based on:
        - neurological level
            - most caudal segment with normal function
        - severity of neurological deficit
            - graded A-E (A complete, E normal) based on degree of intact motor and sensory function
        - an ASIA chart should be used
    - examination and documentation should be performed serially
        - allows early detection of injury progression or of resolution of spinal shock
            - spinal shock is a flaccid areflexia that may occur after spinal cord injury and may last hours to weeks
            - resolves as soft tissue swelling improves
- c-spine precautions for trauma patients
    - neck pain or neurological symptoms
    - altered level of consciousness
    - significant blunt injury above the level of the clavicles
- immobilisation
    - hard collar is to remind patient and healthcare workers that the neck has not been cleared
    - requires additional manual immobilisation or sand bags/blocks and tape
- approach to patient with spinal cord injury
    - trauma team activation
    - transfer patient from rigid board to trauma mattress as soon as possible
    - primary survey
        - airway with c-spine immobilisation
            - lesions above C5 require intubation for mechanical ventilation
            - maintain cervical spine precautions
                - hard collar
                - sandbags/blocks
                - tape forehead
        - breathing and ventilation
            - oxygen 15l/min via non-rebreather mask
            - monitor for respiratory insufficiency from thoracic or higher spinal lesions, complications of spinal cord injury or other thoracic injuries
            - respiratory insufficiency
                - causes vary according to level of injury
                    - high cervical injuries may lead to airway obstruction due to local haematoma and swelling
                    - lesions at C5 or higher lead to diaphragmatic paresis or paralysis (phrenic nerve arises from C3-C5
                    - thoracic or higher lesions may lead to respiratory distress due to paralysis of intercostal muscles (intercostal nerves arise from T1-T12)
                - other causes include:
                    - coexistent thoracic injuries

                - coexistent traumatic brain injury (e.g. decreased respiratory drive)
                - complications of spinal cord injury (e.g. aspiration, atelectasis, pulmonary embolus, metabolic acidosis from spinal shock)
                - complications of treatment (e.g. sedation, fluid overload, transfusion associated lung injury, ventilator associated pneumonia)
        - circulation with haemorrhage control
            - look for neurogenic shock
                - may mask haemorrhagic shock, which is more common
                - characterised by hypotension, bradycardia and peripheral vasodilatation
                - occurs after a significant proportion of the sympathetic nervous system has been damaged – may occur with lesions at T6 or higher
                - treat with repeated fluid boluses (e.g. 250ml crystalloid) +/- noradrenaline infusion to maintain organ perfusion
                    - aim for normal mentation, warm peripheries, urine output >0.5ml/kg/h, MAP >65mmHg
                - be aware that vagal stimuli (e.g. suction, NG tube insertion, intubation) may exacerbate neurogenic shock
                    - treat with atropine 600 microgram IV boluses
        - disability
            - detailed neurological examination including motor and sensory levels bilaterally
            - check for priapism
            - check anal sphincter tone and bulbocavernosus reflex
        - exposure and environment
            - higher risk of hypothermia due to peripheral vasodilation resulting from sympatholysis and decreased muscular activity
            - use fluid warmer, warm blankets and/or bair hugger
    - adjuncts to primary survey and resuscitation
        - ECG and full non-invasive monitoring including temperature
        - trauma CT
        - FAST scan
        - NG tube
            - spinal patients are at risk of gastric distension
        - urinary catheter
            - prevent retention and bladder distension
            - monitor urine output
    - transfer
        - consider early transfer to spinal unit
    - secondary survey
        - head to toe examination
    - adjuncts to secondary survey
        - may include MRI

        - monitor for fluid overload using bedside ultrasound (IVC distension, pulmonary oedema)
    - ongoing critical care
        - FAST HUGS IN BED, PLEASE gives things to consider
            - **f**luid therapy and feeding
            - **a**nalgesia, antiemetics
            - **s**edation and spontaneous breathing trial
            - **t**hromboprophylaxis
            - **h**ead up position (30 degrees) if intubated
            - **u**lcer prophylaxis
            - **g**lucose control
            - **s**kin/eye care and suctioning
            - **i**ndwelling catheter
            - **n**asogastric tube
            - **b**owel care
            - **e**nvironment (e.g. temperature control, appropriate surroundings in delirium)
            - **d**e-escalation (e.g. end of life issues, treatments no longer needed)
            - **p**sychosocial support (for patient, family, staff)
- investigations
    - CT within one hour if head injury and any of the following:
        - GCS <13
        - patient is intubated
        - plain x-rays abnormal or technically inadequate
        - definitive diagnosis required (e.g. before surgery)
        - patient is alert and stable and there is clinical suspicion of cervical injury with any of:
            - age ≥65
            - dangerous mechanism of injury
                - e.g. fall >1m
                - fall down 5 stairs
                - axial load to head
            - focal neurological deficit
            - paraesthesia in the upper or lower limbs
        - other areas are also to be scanned (e.g. multi-level trauma)
    - imaging generally required if:
        - point tenderness
        - deformity or bony step
        - neurological findings consistent with a thoracic or lumbar injury
        - high risk mechanism, especially in the presence of distracting injuries
- cervical spine injuries
    - pathophysiology
        - anatomy
            - bony components
                - vertebral body which give the vertebra its strength and weight bearing ability
                - vertebral arch formed by pedicles and laminae

        - vertebral foramina which align to form the vertebral canal for the spinal cord
        - seven processes
            - spinous process projects posteriorly from the vertebral arch
            - two transverse processes posterolaterally from the intersection of the pedicles and laminae
                - the spinous and transverse processes allow attachment of the deep muscles of the back
            - four articular processes, two superior and two inferior
                - articulate with the vertebrae above and below
    - ligamentous components
        - anterior longitudinal ligament
            - strong, broad fibrous band which covers the anterior of the vertebral bodies and intervertebral discs and extends from the pelvic surface of the sacrum to the anterior tubercle of C1
            - maintains stability of the intervertebral joints and prevents hyperextension of the vertebral column
        - posterior longitudinal ligament
            - narrower and weaker fibrous band which runs within the vertebral canal along the posterior aspects of the vertebral bodies from C2 to sacrum
            - helps prevent hyperflexion of the vertebral column and protrusion of the intervertebral discs
        - the ligamentum flavum
            - joins the laminae of adjacent vertebral arches; binds the laminae of adjacent vertebrae together and forms part of the posterior of the vertebral canal
            - resists separation of the vertebral laminae and attempts to prevent abrupt flexion of the vertebral column
        - the interspinous and supraspinous ligaments
            - adjacent vertebrae are joined by weak interspinous and strong supraspinous ligaments
            - the supraspinous ligament merges with the nuchal ligament in the neck
        - the intertransverse ligaments
            - connect adjacent transverse processes with scattered fibres in the cervical spine, becoming stronger in the thoracic spine
- mechanism of injury
    - hyperflexion
        - causes compression of the anterior aspects of the vertebral bodies and posterior ligament complex disruption
        - leads to:

        - Chance fractures
            - horizontal fracture through the body, pedicle and posterior elements of the vertebrae
        - tear drop fractures
            - fracture of the anterior superior corner of inferior vertebrae
        - rupture of posterior ligament
    - odontoid peg
        - may be fractured by sudden severe flexion
- flexion and rotation
    - 50-80% of cervical injuries are caused by this mechanism as are most thoracolumbar injuries
    - mechanism causes disruption of the posterior ligament complex and the posterior column
    - the facet joints, lamina, transverse processes and vertebral bodies may fracture
    - relatively flat facet joints may dislocate without fracture
    - spinous processes of C6/7 can also be avulsed by interspinous ligaments
        - called clay-shoveller's fracture
    - all intervertebral ligaments may tear and the upper vertebral body can be displaced relative to the one below
- hyperextension
    - damages the anterior column
    - associated with anterior fracture of the anterior inferior aspect of the vertebral body
    - the posterior aspect of the vertebral body may be crushed, with a risk of retropulsion of bony fragments or the intervertebral disc into the spinal canal
    - hangman's fracture:
        - hyperextension fracture through the pedicles of C2 following hyperextension with distraction or compression
        - results from judicial hanging (rather than suicidal which causes asphyxiation) or from striking the chin on the steering wheel in a collision
- rotation
    - rarely occurs in isolation
    - primary injury occurs to the posterior ligament complexes, and is often unstable
    - may result in facet joint dislocation
- compression
    - common in thoracic and lumbar spine injuries
    - results in wedge fractures
    - Jefferson fracture
        - C1 fracture cause by an axial loading mechanism
            - e.g. weight landing on patient's head or patient landing on their head after a fall

        - the atlas is compressed between the occipital condyle and C2
        - the laminae and pedicles are fractured and the transverse ligament holding the peg in position can be torn
        - the skull and C1 may slide forward on C2
        - there can be significant shift before compression of the spinal cord occurs as 1/3 of the space of the spinal canal is occupied by the odontoid peg, 1/3 by the areolar tissue and 1/3 by the spinal cord itself
    - differences between adults and children
        - children have relatively larger heads
        - their ligaments and joint capsules are more lax
        - their facet joints are more horizontal
        - their vertebral bodies are wedge shaped
        - consequences of this are:
            - pseudosubluxation refers to the appearance of forward slippage of one vertebral body on another
                - pseudosubluxation of C2 on C3 occurs in 24% of under 8 year olds and of C3 on C4 in 14% of under 8 year olds
            - SCIWORA (spinal cord injury without radiological abnormality
                - objective signs of myelopathy as a result of trauma with no evidence of fracture or ligamentous instability on plain radiographs or tomography
                - more common in children and reported to occur in up to 30% of spinal cord injuries in children
            - younger children under 10 years are more likely than older children to:
                - injure the upper cervical spine (C1-C4) compared with the lower c-spine
                - dislocate the cervical spine
                - injure the spinal cord itself
            - the cervical spine takes on its adult form from about the age of 8 years
    - clinical assessment – adults
        - conscious co-operative patients
            - most commonly encountered group
            - low incidence (<3%) of c-spine injury
            - focused history and examination
            - clinical decision rules exist:
                - NEXUS low risk criteria
                    - low risk of injury if all 5 criteria met:
                        - no midline cervical tenderness
                        - no focal neurological deficit
                        - normal alertness
                        - no intoxication
                        - no painful distracting injury

        - negative predictive value 99.8% (and low specificity – 12.9%, so most patients with positive criteria still did not have actual injury
    - Canadian c-spine rules
        - asks 3 questions:
            - is there any high risk factor that mandates radiography?
                - age ≥65 OR
                - OR dangerous mechanism
                    - fall from ≥1 metre/5 stairs
                    - axial load to head (e.g. diving)
                    - RTC high speed (≥60mph), rollover, ejection
                    - motorised recreational vehicles
                    - bicycle collision
                - OR paraesthesias in extremities
            - is there any low risk factor that allows safe assessment of range of motion? (if no, imaging required)
                - simple rear end RTC
                    - excludes:
                    - pushed into oncoming traffic
                    - hit by bus/large truck
                    - rollover
                    - hit by high-speed vehicle
                - OR sitting position in ED
                - OR ambulatory at any time
                - OR delayed onset of neck pains
                - OR absence of midline c-spine tenderness
            - are they able to actively rotate the neck (45 degrees left and right)?
                - if yes, no imaging required
                - if no, imaging required
        - sensitivity is 100%, specificity 42.5%
    - unconscious/unco-operative patients
        - cannot have their c-spine cleared clinically
        - require imaging
- clinical assessment in children
    - there is no robust evidence base for a clinical rule-out for cervical spine injury in children under 10
- investigations
    - plain cervical spine series
        - x-rays do not detect all c-spine fractures

- imaging consists of three views: lateral, AP and odontoid peg
    - in children under 5 the peg view is considered unnecessary
- lateral film
    - adequacy
        - visible base of skull to top of body of T1
    - alignment
        - along anterior margins of vertebral bodies
        - posterior margins of vertebral bodies
        - bases of spinous processes
        - bone inspection of vertebral bodies
        - intervertebral discs – uniform height
        - soft tissues:
            - C1-4 max 7mm (30% of vertebral body width)
            - C5-7 max 22mm (100% of vertebral body width)
        - peg
            - C1 no more than 3mm in adults (5mm in children) space between anterior arch of C1 and peg
- AP view
    - spinous processes should be in a straight line except if bifid spinous process
    - distance between spinous processes approximately equal
- odontoid peg view
    - lateral margins of C1 should lie within lateral margins of C2
    - spaces on each side of peg should be equal – slight variation if neck is slightly rotated
- if lateral views are inadequate:
    - arm pull view in which the patient's arms are pulled down to try and lower the shoulders so the lower cervical spine can be visualised
    - swimmer's view – the arm nearest the x-ray is elevated and the arm nearest the plate is kept extended
        - can be difficult to interpret due to overlying bones
- flexion/extension views should not be done – they do not add anything in conscious patients and risk further damage in unconscious patients

- CT
    - indication should be carefully considered as patients undergoing CT of the whole cervical spine have a 14-fold increase in the dose of radiation to the thyroid compared to 3 image radiographs
    - indications as primary modality:
        - GCS below 13 on initial assessment
        - patient has been intubated
        - plain film series technically inadequate (e.g. desired view unavailable), suspicious or definitely abnormal

        - continued clinical suspicion of injury despite a normal x-ray
        - patient is being scanned for multi-region trauma
    - MRI
        - very sensitive for soft tissue injuries, including ligament injuries, disc herniation and haemorrhage, which are less well visualised on CT
            - many will not be clinically significant but a minority represent unstable injuries
        - it is less sensitive than MRI for imaging the posterior elements of the spine and the craniocervical junction
        - indicated if there is neurology referrable from the c-spine or if there is severe pain with a normal CT and concern about ligamentous injury
        - first line in children if there is a strong suspicion of c-spine injury
            - plain x-rays can be considered if they do not meet the criteria for MRI but clinical suspicion remains after repeated clinical assessment
                - findings should be discussed with a consultant radiologist and further imaging done if necessary
- management
    - neck sprain
        - analgesia
        - mobilisation
        - avoid collar immobilisation
    - spinal cord injury
        - keep immobilised
        - immediate discussion with on call neurosurgeon or spinal surgeon by the trauma team leader or senior doctor
- prognosis
    - neck sprain
        - most patients have a good prognosis
        - risk factors for worse prognosis include:
            - older age
            - female gender
            - baseline neck pain and headache intensity
            - baseline radicular signs/symptoms
            - insurance and compensation claims
                - instruction of a lawyer shortly after a collision is independently associated with slower recovery

TP3 chest and lung injury

- tension pneumothorax
    - background
        - an expanding pneumothorax that increasingly limits ventilation and venous return
        - even impressive expansion may be well tolerated in young individuals with no comorbidities or other injuries
    - clinical assessment and identification

        - awake patients
            - chest pain and respiratory compromise
            - low saturations may be an early feature
            - hypotension tends to be a late feature
            - lateralising may not be straightforward
                - decreased breath sounds may be easier to listen for in the axillae
            - the classical signs of hyper-resonance and tracheal deviation are difficult to elicit
        - ventilated patients
            - early reliable signs
                - decrease in oxygen saturations (likely prompt)
                - decrease in BP
                - tachycardia
            - raised ventilation pressure (>40)
                - ensure pressure alarms are set appropriately
    - investigation
        - if critically unwell, treatment should not be delayed
        - POCUS
            - absence of lung sliding
        - CXR
            - can be done if patient is not unstable and there is diagnostic uncertainty
            - radiological evidence of tensioning does not necessarily correlate clinically
    - management
        - needle thoracocentesis
            - advocated in the first instance in the ATLS manual
            - drawbacks include:
                - tends to be overused, particularly in stable patients where portable x-ray is available and chest drain the preferred treatment
                - lack of hiss (or bubbling if saline in a syringe attached to the needle) may be considered evidence of no pneumothorax but does not have 100% sensitivity
                - a standard 14G cannula may not reach the pleural space via the second intercostal space
                    - ATLS guidelines now recommend needle decompression via the 4^{th} or 5^{th} intercostal space anterior to the mid axillary line
                - the cannula can kink and cease to function
                - a pneumothorax can be caused if the diagnosis is wrong (of particular concern if the patient requires IPPV)
            - in peri-arrest patients with suspected tension pneumothorax (particularly if penetrating trauma), a thoracostomy is probably the best option
        - chest drain

                - the most common cause of serious injury (and death) is insertion at the incorrect site, usually too low)
                - confirm that the drain lies in the chest wall cavity by looking for fogging of the tube and swinging of the chest drain with respiration
                - the tube should not be clamped and suction should not be applied
                - the underwater seal must remain below the insertion site at all times
            - tips
                - have saline in the syringe if performing needle thoracocentesis to confirm placement
                - gross surgical emphysema in combination with pneumomediastinum and a chest drain that continues to bubble suggests tracheo-bronchial injury
                - if there is good clinical and radiological evidence of significant lateral chest wall injury, consider the second intercostal space anteriorly for chest drain insertion
                    - safer for the operator and less painful for the awake patient
            - pitfalls
                - a third of initial CXRs in trauma do not detect pneumothorax
                    - important for anaesthetists to be aware of this if the patient goes to theatre
                - cardiac tamponade can give similar clinical signs, with shock and distended neck veins
                    - a combination of POCUS, CXR and mechanism of injury should help to distinguish
                - other pathology may masquerade as pneumothorax on CXR
                    - e.g. emphysematous bulla, gastrothorax
                    - consider CT if the diagnosis remains in doubt
- open pneumothorax
    - background
        - rare, typically caused by ballistic injury and unlikely to be missed clinically
        - as the patient breathes in, the hole in the chest competes with the normal airway for delivery of air
            - a hole of only 1-2cm diameter can cause significant compromise, particularly where there are other comorbidities or significant injuries
    - clinical assessment and identification
        - examination of front and back of chest
    - management
        - definitive treatment is surgical repair
        - cover the wound
            - Jelonet or chest seal (Ashermann, Bolin)
            - if the hole is too big, cover with a large opsite, make a small hole in the middle and place a chest seal (if available) on top
            - if a dressing is used it should not be occluded on all four sides to allow for a flutter valve effect

            - if there is suspicion of tensioning, the dressing should be temporarily released
        - early intubation
            - solves the respiratory embarrassment created by the hole in the chest
        - for small open pneumothoraces, a chest drain should be inserted remote from the wound on that side
            - easier once the patient is anaesthetised
        - a chest drain should not be inserted for patients with a large open pneumothorax as muscle flaps may be needed for closure and can be damaged in the procedure
- massive haemothorax
    - background
        - defined as a haemothorax of >1500ml, or greater than one third of the patient's blood volume
        - uncommon, may be caused by blunt or penetrating trauma, unlikely to be missed radiologically
        - creates a problem because of shock (haemorrhagic and impaired venous return from the vena cava) and decreased ventilation (due to compression of the lung on that side)
    - clinical assessment and identification
        - auscultation of lung bases
        - signs of shock
        - POCUS/CXR
            - on POCUS, absence of a mirror image of liver/lung or spleen/lung across the diaphragm suggests haemothorax
                - free fluid in the abdomen alone should prompt reconsideration of the diagnosis
            - size of haemothorax may be underestimated on supine CXR
    - management
        - focused on simultaneously restoring blood volume and decompressing the chest
        - use blood and blood products early, with activation of major haemorrhage protocol if required
        - uncrossmatched blood may be required
            - ATLS recommends no more than 1 litre of crystalloid as a temporising measure until blood available
        - 28-32F chest drain to decompress the chest and improve ventilation
        - ideally the drain should be connected to a cell salvage/saver machine
            - if unavailable, the usual equipment should be used, but the underwater seal should be primed with saline not sterile water – the saline/blood can still be run through a cell saver later
        - >1500ml of blood immediately returning from the chest drain is generally an indication for urgent thoracotomy
            - cardiothoracic surgeons should be involved early
        - occasionally a massive haemothorax can be well tolerated, typically in young patients with a chest stabbing

                - it may be appropriate to delay chest drain insertion until the patient is in theatre where there is cell salvage available
            - ATLS indications for thoracotomy:
                - prompt drainage of 1500ml blood or a third of the patient's circulating volume
                - >200ml/h blood loss for 2-4 hours
                - continued need for blood transfusion
- cardiac tamponade
    - background
        - collection of fluid in the pericardial sac causing haemodynamic compromise
        - progression to PEA arrest may be rapid
            - 50-100ml of blood in the sac may be enough
        - cardiac tamponade as a result of blunt injury is exceptionally rare in patients reaching hospital alive
    - clinical assessment and identification
        - FAST has high sensitivity (around 95%)
        - there are drawbacks in detecting and interpreting the classical clinical signs
            - neck veins may not be distended if the patient has haemorrhagic shock
            - hypotension and raised respiratory rate may have other causes
            - muffled heart sounds are unlikely to be heard in the ED
    - management
        - fluid resuscitation to increase preload
        - if haemodynamically stable, refer for urgent surgical exploration in theatre
            - look for co-existing injuries such as pneumothorax
        - thoracotomy if patient presents within 10 minutes of cardiac arrest
            - pericardiocentesis even if correctly performed is unlikely to succeed as the blood will be clotted
                - the procedure will also delay thoracotomy
                - it should only be attempted if there really is nobody capable of opening the chest
- flail chest
    - background
        - a series of ribs (usually 3 or more) fractured segmentally (i.e. in more than one place) resulting in a free or floating section of the chest wall
        - relatively common, and small flails may be missed clinically
        - underlying pulmonary contusions are inevitable but may cause significant morbidity and mortality
        - considerable force is required to cause a flail chest so other injuries should be sought in young people
        - multiple rib fractures are a potential source of significant haemorrhage
    - clinical assessment and identification
        - palpation and inspection
        - CXR
            - associated pneumothorax, haemothorax, pulmonary contusions
                - appearance of early pulmonary contusions is particularly worrying as more may evolve

- management
  - depends on level of compromise
  - consider:
    - patient's clinical condition
    - size of flail chest
    - associated injuries
    - age
    - co-morbidities
    - destination from resus (theatre, CT scan, ITU, ward)
  - for patients with major trauma:
    - intubation and ventilation (intermittent positive pressure ventilation)
      - allows better control of respiratory compromise
      - address pain with adequate analgesia post-RSI
      - can facilitate clinical procedures such as CT scan and chest drain insertion
  - chest drain for associated haemothorax and pneumothorax
  - careful fluid resuscitation
    - excessive fluid floods injured lung tissue
  - definitive surgery (internal fixation of ribs) may be considered by cardiothoracic surgeons
  - a conservative approach may involve the use of thoracic epidural, intercostal nerve blocks and/or patient controlled analgesia, CPAP and physiotherapy

- pulmonary contusion
  - background
    - bruised lung
    - unlikely to be missed radiologically unless CXR is done early
    - potentially life-threatening since:
      - patient is at risk of hypoxaemia
      - because of the force involved to cause the injury, associated injuries are common
      - injured lung is vulnerable to flooding from excessive fluid resuscitation
    - patients with co-morbidities and/or advanced age are particularly at risk
  - clinical assessment and identification
    - CXR
      - patchy white areas progressing to frank consolidation
      - aspiration and haemorrhage are differentials
      - contusions visible on the initial CXR suggest significant injury, with further radiological changes and blood gas derangement likely to follow
      - look for associated rib fractures +/- haemo/pneumothorax
      - rib fractures do not always co-exist, particularly in the young
        - their existence suggests there was significant force
  - management
    - IPPV with PEEP for sicker patients
    - judicious use of fluids with consideration of central line and arterial line

        - no evidence for steroids or prophylactic antibiotics
        - disposition should be discussed with ITU and thoracic surgical colleagues
- myocardial contusion
    - background
        - myocardial bruising caused by blunt injury, including deceleration and ballistic mechanisms
        - there is a lack of diagnostic gold standard
    - clinical assessment and identification
        - normal ECG effectively rules out
            - beware labelling ST changes as contusion as a primary cardiac event may have precipitated an accident
        - unexplained tachycardia may be a clue
            - also look for atrial and ventricular ectopics
        - consider bedside echo
        - consider troponin
    - management
        - no direct ED-based intervention
        - treat the following if identified:
            - hypoxaemia
            - acidaemia
            - fluid status
            - low haemoglobin
        - monitor ECG and consider central line and arterial line
- aortic injury
    - background
        - commonly missed clinically and on CXR
        - consider in rapid deceleration injuries (e.g. RTA, fall)
        - around 10% survive to the ED
            - they have a haemorrhage tamponaded by aortic adventitia (pseudo-aneurysm)
        - no increased risk related to atherosclerosis
        - young males are the most likely due to risky behaviour
    - clinical assessment and identification
        - most patients do not have specific symptoms
        - the majority of ruptures reaching hospital alive are just distal of the left subclavian artery so difference in blood pressure between arms is not necessarily a feature
        - occasional clinical clues include:
            - generalised hypertension
            - upper extremity hypertension in combination with weak or absent femoral pulses
            - harsh systolic murmur
        - CXR features
            - widened mediastinum
                - most reliable sign
            - fractures of the first and second ribs
            - obliteration of the aortic knob

                - deviation of the trachea to the right
                - presence of a pleural cap
                - elevation and rightward shift of the right main stem bronchus
                - depression of the left mainstem bronchus
                - obliteration of the space between the pulmonary artery and the aorta
                - deviation of a nasogastric tube to the right
            - contrast CT thorax is the investigation of choice
        - management
            - surgical repair or endovascular stenting
            - competing and likely overriding injuries include life-threatening head, abdominal or pelvic injuries
            - control hypertension and tachycardia
                - systolic no more than 110mmHg
                - labetolol is the agent of choice
- diaphragmatic injury
    - background
        - usually caused by penetrating injury
        - easily missed clinically and radiologically
        - in blunt injury it is three times more likely on the left (the right hemi-diaphragm is protected by the liver) and nearly always at the weakest point, posterolaterally
        - a diaphragmatic breach will not heal spontaneously because of the differential pressure gradients between chest and abdomen
        - abdominal content herniation can occur even years later
    - clinical assessment and identification
        - symptoms are likely to be masked by associated injuries
        - diaphragm injuries resulting from knives or bullets are more likely to be detected on surgical exploration
        - in blunt injuries (particularly those causing an abrupt rise in intra-abdominal pressure) it is important not to misinterpret a gastrothorax as a pneumothorax
    - management
        - gentle insertion of NG tube to drain stomach content
            - if there is diagnostic uncertainty between gastrothorax and pneumothorax, NG placement will help with confirmation
        - a cautiously placed chest drain (using the open method) may be indicated, with care to avoid abdominal contents
        - surgical repair needs to be considered in the context of related injuries
- oesophageal injury
    - rare
    - often initially missed clinically and radiologically
    - other associated injuries are likely to predominate
    - operative repair or endoluminal stenting will need to be considered
- trachea-bronchial injury
    - background
        - rare

        - caused by significant deceleration injuries
        - most patients die at the scene
        - unlikely to be missed in survivors as the effects are dramatic
    - clinical assessment and identification
        - massive air leak suggested by:
            - gross surgical emphysema
            - pneumomediastinum
            - vigorously bubbling chest drain that has failed to alleviate respiratory compromise
        - haemoptysis is an additional clue
    - management
        - intubation strategy should be discussed with senior anaesthetic colleagues (e.g. single or double cuffed tubes, use of fibreoptics)
        - consider additional large bore chest drain on the affected side, one intercostal space further up
            - do not attach suction to the chest drain
        - other significant injuries may influence the resuscitation strategy
- simple pneumothorax
    - common injury, often missed on CXR and subsequently found on CT
    - small, asymptomatic pneumothoraces may be observed, even if the patient is ventilated
        - around a third may deteriorate clinically and require a drain
    - there is no guideline for the safe time for flying following a simple traumatic pneumothorax
        - a pragmatic approach may be to use the guidelines for spontaneous pneumothorax and allow flying once CXR confirms resolution of the pneumothorax
- rib fractures
    - significant force required in young people
        - underlying injury is typical, especially lung contusions
    - less force is required in the elderly but even an isolated rib fracture can result in severe morbidity (e.g. secondary pneumonia), particularly in those with co-morbidities
        - in addition to standard therapy, consider patient-controlled analgesia, thoracic epidural and physiotherapy for vulnerable patients
- sternal fractures
    - relatively benign injuries but may be associated with underlying myocardial or pulmonary contusion
    - depending on mechanism, screening for neck injury should also be done
    - in vulnerable patients, consider patient-controlled analgesia or local anaesthetic via a sternal catheter
- posterior sternoclavicular joint dislocation
    - exceptionally rare
    - clinically important because the medical clavicular head may compromise the airway or major vessels
    - if there is evidence of compromise, reduction of the dislocation should be attempted

        - abduct the arm to 90 degrees, extend 10-15 degrees and apply traction (with counter traction to the torso)
        - maintain traction and pull the medial end of the clavicle forward with fingers and thumb
        - if this fails, prepare the skin with iodine and local anaesthetic and repeat with a towel clip

TP4 major vascular injury

- types
    - extremity
        - injury to the vasculature of the arms or legs
    - junctional
        - vascular injury where the extremity meets the torso (e.g. hip, axilla, base of neck)
    - non-compressible truncal
        - anywhere on the torso involving large vasculature
    - subtypes include:
        - occlusive
            - transection
            - thrombosis
            - embolism
        - reversible spasm
        - non-occlusive
            - laceration
            - intimal flap
            - pseudoaneurysm
            - A/V fistula
            - compartment syndrome
    - occult upper extremity vascular injury
        - clavicle/first rib fracture
            - subclavian artery
        - anterior shoulder dislocation
            - axillary artery
        - proximal humerus fracture
            - axillary artery
        - humeral shaft fracture
            - brachial artery
        - elbow dislocation
            - brachial artery
- clinical features
    - hard signs
        - absent distal pulses
        - signs of distal ischaemia
            - pain
            - pallor
            - paraesthesia
            - paralysis

                - poikilothermia
            - audible bruit or palpable thrill at injury site
            - active pulsatile haemorrhage
            - large expanding haematoma
        - soft signs
            - small nonexpanding haematoma
            - subjectively decreased pulse
            - peripheral nerve deficit
            - history of pulsatile or significant haemorrhage at time of injury
            - unexplained hypotension
            - high risk orthopaedic injuries (fracture, dislocation, penetration)
- investigation
    - Arterial Pressure Index (API)/Injured Extremity Index (IEI)
        - doppler determined arterial systolic blood pressure in the injured limb divided by the systolic blood pressure in the uninjured limb
        - <0.9 abnormal, >0.9 highly sensitive for excluding major vascular injury
        - allows for serial, objective monitoring
        - only detects obstructive lesions
        - unreliable in proximal injuries, popliteal injuries, shotgun wounds, multiple wounds, shock
        - false negative with deep femoral artery injury
    - imaging
        - CT angiography
            - gold standard for excluding vascular injury
            - highest sensitivity and specificity
            - useful for detection of other injuries (venous, neural, fractures etc.)
        - duplex Doppler
            - operator dependent, does not definitively exclude arterial injury
            - sensitive for vessel injury, thrombosis, pseudoaneurysm, intimal flap and A-V fistula
        - POCUS
            - useful as an adjunct, no randomised controlled trials proving sensitivity
    - evaluation algorithm
        - hard signs
            - >90% risk of arterial injury
            - 50% require intervention
            - requires immediate arterial exploration without further investigation
        - soft signs
            - 30% risk of arterial injury
            - perform API – if <0.9, admit for serial API
            - consider:
                - doppler US
                - CT angiogram
                - evaluation of compartment pressures
- extremity trauma

- recognition
    - penetrating extremity injury (e.g. stab or gunshot) or severe blunt trauma (e.g. arterial injury due to associated fracture)
    - cold, pale and pulseless distal extremity or rapidly expanding haematoma suggests arterial compromise
    - check arterial pressure index
    - assess for haemorrhagic shock
    - angiography only if the patient is stable
- management of severe arterial haemorrhage
    - immediate surgical review
    - apply direct pressure and elevation
        - direct digital pressure
            - PPE
            - ensure no hazardous objects in wound
            - use one finger with interposed gauze to press directly on the bleeding vessel just proximal to the bleeding point
            - maintain for 10 minutes
    - consider applying adrenaline soaked gauze or haemostatic dressings if available
    - pressure bandage
        - 'nugget' method
            - substitute the occluding finger with a dental roll or tightly folded 'nugget' of gauze
                - a temporary proximal tourniquet may facilitate this
            - once the positioning is correct and there is no further bleeding, slightly larger or less folded pieces of gauze can be applied one on top of the other to create an inverted pyramid of gauze
            - the layers of gauze are secured with a loose bandage; only very light pressure is needed as the pressure is focused on the bleeding point (based on the equation pressure =force/area)
            - the tightness of the bandage can be judged from the amount of pressure needed to maintain haemostasis when applying the top layer of gauze
    - tourniquets may be life saving
        - easiest way in the ED is a blood pressure cuff proximal to the bleeding point
            - inflate the cuff above systolic pressure
            - clamp the tubing with a haemostat to prevent leakage and loss of pressure
        - an alternative is a pneumatic cuff, like used for a Bier's block
        - when applying a tourniquet:
            - record the time of application
            - perform a neurological exam at the time of application
            - do not leave the tourniquet on for more than 120 minutes
    - reduce and splint long bone fractures, apply a pelvic binder for pelvic fractures

        - correct coagulopathy and commence haemostatic resuscitation as required
        - do not clamp or tie off a vessel unless it is superficial and clearly visible
            - blindly clamping an artery may damage a nerve running alongside it
    - surgical treatment options
        - injuries to most major 'named' arteries requiring repair or intervention include:
            - extravasation
            - pulsatile haematoma
            - occlusion
            - pseudoaneurysm
            - fistula formation
        - surgical repair options include:
            - direct repair
                - sutures
                - patch angioplasty
                - interposition graft
                - vein patches
            - ligation
                - only small, distal and redundant arteries
            - damage control surgery
                - uses intravascular shunts to allow immediate restoration of distal blood flow, with later definitive repair once the patient has been resuscitated and normal physiology restored
        - interventional radiology measures such as embolization are useful in certain arterial injuries
        - injuries that do not normally require immediate repair include:
            - injuries such as intimal defects
                - 87-95% heal spontaneously
            - some arteries (profunda femoris, anterior tibial, posterior tibial, peroneal) do not require surgery but can be reimaged at 3-5 days to check progress if occluded, or undergo embolization if the injury involves extravasation or arteriovenous fistula
- aortic and great vessel injury
    - background
        - aortic disruption in trauma usually involves a tear on the aortic wall due to acceleration-deceleration forces
        - may result from penetrating (usually die at scene) or blunt trauma
        - those who make it to hospital may only have the outer aortic wall (adventitia) intact, containing a haematoma
    - recognition
        - conscious patients may have tearing chest and back pain
            - neurological deficits may also be present (e.g. dissection involving origins of carotid arteries, spinal arteries etc.)
            - clinical signs (e.g. differences in pulses and blood pressure between the upper limbs) are unreliable

        - suspect based on mechanism and the presence of other injuries (e.g. fractures of sternum, upper ribs, scapula)
        - look for features of aortic dissection on CXR (especially widened mediastinum) – features are often absent
        - low threshold for CT angiogram
        - pericardial tamponade or aortic dissection flap may be seen on echo
    - management
        - high flow oxygen via non-rebreathe mask
        - avoid excessive fluid resuscitation
        - lower the pulse rate to decrease aortic shear forces by commencing beta blockade infusion, then commence GTN infusion to aim for systolic 90-100mmHg and adequate tissue perfusion
        - definitive treatment is surgery, stenting or both

TP5 abdominal injury

- background
    - assessment of abdominal trauma is difficult
    - serious injuries may appear to be innocuous and localisation of symptoms is variable
    - a high index of suspicion is required to recognise occult injuries
    - associated injuries can further complicate assessment
    - penetrating trauma
        - injuries are direct as the penetrating item passes through organs and tissues
        - most commonly knife-related in the UK
            - the wound may be small and insignificant in appearance
            - information on the site of entry, direction of injury and the size and nature of the penetrating item is helpful
        - stab wounds and low energy gunshot wounds cause tissue damage by lacerating and tearing
        - high energy gunshot wounds transfer more kinetic energy, causing increased damage surrounding the track of the missile due to temporary cavitation
        - wound assessment
            - patients with penetrating injury, even with normal vital signs, should be assessed by a senior surgeon
                - ideally exploration should be in theatre
            - unless the injury is trivial, patients should be admitted for observation even if no further investigation is initially required
            - objects remaining in the wound should not be removed as they may be tamponading underlying vessels
        - common sites of injury
            - around 50% of penetrating abdominal injuries damage intraperitoneal structures
            - the peritoneal cavity is not breached in 25% of cases
            - stab wounds traverse adjacent abdominal structures and most commonly involve the liver (40%), small bowel (30%), diaphragm (20%) and colon (15%)
    - blunt trauma
        - commonly results from either a compression force or a deceleration injury

        - greater mortality than penetrating injury as there is often injury to multiple organ systems
        - there are a number of classic injury patterns which occur following common mechanisms such as rapid deceleration whilst wearing a lap belt or after direct impact with a car steering wheel
        - the spleen and liver are the most commonly injured intraperitoneal organs in blunt trauma
        - injury to intraperitoneal structures should be considered when evaluating any injury between nipple and pelvis
- pathophysiology
    - the abdominal contents can be divided into:
        - intra-abdominal structures
        - retroperitoneal structures
        - pelvic structures
    - potential injuries
        - lower chest
            - liver
            - spleen
            - diaphragm
            - stomach
        - anterior abdomen
            - liver
            - spleen
            - colon
            - bladder
            - stomach
            - pancreas
            - transverse colon
            - ileum
            - jejunum
        - flank
            - kidneys
            - ureters
            - ascending colon
            - descending colon
        - posterior abdomen
            - great vessels
            - duodenum
            - pancreas
            - spinal cord
- signs and symptoms
    - usually relate to blood loss
        - signs of shock may be present but present late in young healthy people
    - liver
        - 15-20% of intra-abdominal organ injuries but up to 50% of mortality
            - 45% have associated splenic injury
        - conservative management is appropriate in 80% of cases

                - surgical intervention is reserved for ongoing and uncontrolled bleeding
            - CT angiography can be used to detect the source of any ongoing bleeding
            - interventional radiology may be used to embolise a bleeding vessel and remove the need for surgery
        - spleen
            - injury is graded according to CT findings and management guided by grade
                - grade 1
                    - minor subcapsular tear or haematoma
                - grade 2
                    - parenchymal injury not extending to the hilum
                - grade 3
                    - major parenchymal injury involving vessels and hilum
                - grade 4
                    - shattered spleen
            - the spleen contains around 1 unit of blood at any time
            - grade 1 or 2 injuries can usually be managed conservatively
        - pancreas
            - injury to the pancreas may cause pancreatitis
                - may develop over days
            - blunt pancreatic injury may not immediately be recognised
                - relatively uncommon (occurs in around 10% of blunt abdominal injuries)
                - rarely an isolated injury due to the position of the pancreas
            - amylase elevation will often not occur until 3-4 hours after injury; lipase is no more specific
        - hollow viscus
            - peritoneal contamination with bowel contents will produce peritonism
            - there may be accompanying blood loss but the degree of hypovolaemia is generally less than in solid organ injury
            - damage to the retroperitoneal portion of the bowel will not produce classical signs of peritonism as the leak will be contained
        - vascular structures
            - catastrophic blood loss may occur with injury to any of the large vessels in the abdomen
            - aortic injury is usually fatal, but may be tamponaded if it occurs retroperintoneally
            - injury to the inferior vena cava is likely to be associated with more insidious blood loss unless there is a large tear
        - GU tract
            - bruising, haematuria or meatal blood are often the only signs of a GU injury
            - injury to the intraperitoneal portion of the bladder may result in chemical peritonitis
- clinical assessment
    - priority is to identify immediately life-threatening injuries
    - early airway protection, ventilatory support and circulatory resuscitation are paramount

    - an accurate history can be helpful
    - clinical examination can be unreliable and falsely reassuring in the multiply injured patient
    - a significant mechanism and injury to adjacent body cavities should be taken seriously and prompt a thorough search for abdominal trauma
    - analgesia
        - significant analgesia may be required prior to abdominal assessment
    - an urgent crossmatch must be sent if there is a suspicion of blood loss in abdominal trauma
- management of haemorrhagic shock
    - the initial recommended IV replacement fluid is 0.9% sodium chloride
    - high flow oxygen, and blood products if indicated, maximise the oxygen-carrying potential of intravascular fluid
    - a patient who is GCS 15 and physiologically normal can be viewed as adequately resuscitated
        - frequent reassessment is required
    - grades of shock
        - grade I
            - <750ml blood loss
            - normal pulse, blood pressure, mental state, respiratory rate
            - urine output 30ml/h
        - grade II
            - 750-1500ml blood loss
            - tachycardic, raised diastolic blood pressure, agitated, normal respiratory rate
            - urine output 20-30ml/h
        - grade III
            - 1500-2000ml blood loss
            - tachycardia, lowered systolic blood pressure, confused, tachypnoeic
            - urine output 5-12ml/h
        - grade IV
            - >2000ml blood loss
            - weak pulse, very low blood pressure, obtunded mental state, tachypnoea
            - urine output <5ml/h
    - fluid resuscitation
        - aggressive fluid resuscitation without adequate haemostasis has been demonstrated to dilute clotting factors and potentially dislodge clots
        - maintaining a normal blood pressure may worsen bleeding
        - if there is ongoing, brisk bleeding, it may be impossible to adequately resuscitate a patient until the tap is turned off
- clinical examination
    - inspection
        - may show evidence of bruising, abrasions or penetrating trauma
        - abdominal distension is suggestive of intra-abdominal haemorrhage and in association with signs of hypovolaemia must be dealt with promptly
    - palpation

        - includes palpation of the renal angles and examination of the external genitalia
        - tenderness or guarding must be further investigated but a multiply injured patient may not report much tenderness even if there is significant injury
        - a rigid abdomen is due to leak of bowel contents into the peritoneum from a hollow viscus injury
        - blood is not a peritoneal irritant by itself and serial examinations may show a gradually distending abdomen with little discomfort
        - rectal examination may be useful
            - may demonstrate bleeding or a high-riding prostate suggestive of a pelvic injury and involvement of the genitourinary tract
            - anal tone and sensation should be tested but cannot be used to rule out pelvic or neurological injury in a patient with head or spinal injuries
    - top to toe examination
        - including log roll
        - actively search for any other injuries
        - hypothermia is associated with significant mortality so should be avoided during exposure
    - types of investigation
        - diagnostic peritoneal lavage
            - advantages
                - early operative determination
                - performed rapidly
                - can detect bowel injury
                - no need for transport from resuscitation area
            - disadvantages
                - invasive
                - risk of procedure-related injury
                - requires gastric and urinary decompression for prevention of complications
                - not repeatable
                - interferes with interpretation of subsequent CT or FAST
                - low specificity
                - can miss diaphragm injuries
            - indications
                - abnormal haemodynamics in blunt abdominal trauma
                - penetrating abdominal trauma without other indications for immediate laparotomy
        - FAST
            - advantages
                - early operative determination
                - non invasive
                - performed rapidly
                - repeatable
                - no need for transport from resuscitation area
            - disadvantages

        - operator dependent
        - bowel gas and subcutaneous air distort images
        - can miss diaphragm, bowel and pancreatic injuries
        - does not completely assess retroperitoneal structures
        - does not visualise extraluminal air
        - body habitus can limit image clarity
    - indications
        - abnormal haemodynamics in blunt abdominal trauma
        - penetrating abdominal trauma without other indications for immediate laparotomy
- CT
    - advantages
        - anatomic diagnosis
        - non-invasive
        - repeatable
        - visualises retroperitoneal structures
        - visualises bony and soft tissue structures
        - visualises extra-luminal air
    - disadvantages
        - higher cost and longer time
        - radiation and IV contrast exposure
        - can miss diaphragm injuries
        - can miss some bowel and pancreatic injuries
        - requires transport from resuscitation area
    - indications
        - normal haemodynamics in blunt or penetrating abdominal injury
        - penetrating back/flank trauma without other indications for immediate laparotomy

- management
    - a definitive management plan should be made as early as possible
        - teams involved may include: surgeons, radiologists, anaesthetists, theatre staff, critical care and transfer teams
    - early trauma CT should be carried out in trauma centres once the patient is haemodynamically stable
        - if the patient remains unstable, senior members of the trauma team (emergency, surgical, anaesthetics) should weigh up the merits of CT vs immediate theatre
    - in a DGH, contacting radiology to arrange imaging and thinking about arranging transfer to a trauma centre should be done as soon as possible
    - patients fall into three categories:
        - those requiring immediate surgery due to catastrophic haemorrhage unresponsive to resuscitation
        - those requiring urgent investigation using appropriate diagnostic tests to determine intra-abdominal injury and decisions on appropriate management
        - those with no abdominal injury on clinical examination with low index of suspicion who require observation and sequential re-examination

- fluid resuscitation
    - up to 1 litre in adults, 20ml/kg in children
    - responses:
        - rapid response
            - vital signs return to normal
            - estimated blood loss is minimal (<15%)
            - need for blood is low
            - type and crossmatch should be done
            - operative intervention may possibly be needed
            - early presence of a surgeon required
        - transient response
            - transient improvement with recurrence of decreased blood pressure and increased heart rate
            - blood loss moderate and ongoing (15-40%)
            - need for blood is moderate to high
            - blood preparation should be type-specific
            - operative intervention is likely
            - early presence of a surgeon is required
        - minimal or no response
            - vital signs remain abnormal
            - estimated blood loss is severe (>40%)
            - need for blood is immediate
            - emergency blood release is required
            - need for operative intervention is highly likely
            - early presence of a surgeon is required

TP6 pelvic injury

- background
    - pelvic fractures are associated with:
        - high energy mechanisms
            - motor vehicle crashes
            - collisions with pedestrians
            - falls from height
        - major haemorrhage
            - can be difficult to control
        - other major injuries
            - intra-abdominal organs (28%) including aortic injury
            - hollow viscus injury (13%)
            - rectal injury (up to 5%)
        - high morbidity and mortality
            - overall mortality 10-30%, up to 50% if shocked
    - stable pelvic fractures that do not involve the pelvic ring (e.g. pubic ramus fracture, avulsion fracture) are associated with much less morbidity
- classification of pelvic fractures
    - two systems often used:
        - Tile classification
            - based on pelvic stability and useful for guiding reconstruction

- Tile A
    - rotationally and vertically stable
    - pubic ramus fracture, iliac wing fracture, pubic stasis diastasis <2.5cm
- Tile B
    - rotationally unstable, vertically stable
    - B1: pubic symphysis diastasis >2.5cm and widening of the sacroiliac joints (open book fracture due to external rotation forces on the hemipelvices
    - B2: pubic symphysis overriding (internal rotation force on hemipelvises)
- Tile C
    - rotationally and vertically unstable
    - disruption of SI joints due to vertical shear forces
    - C1: unilateral
    - C2: bilateral
    - C3: involves acetabulum

- Young-Burgess classification
    - more useful in the ED as based on mechanism and also indicates stability
    - anteroposterior compression (APC)
        - typically result from head on collisions
        - common features is diastasis of the pubic symphysis or vertical fracture of the pubic rami
            - APC I: pubic symphyseal diastasis <2.5cm, no significant posterior ring injury (stable)
            - APC II: pubic symphyseal diastasis >2.5cm, tearing of anterior sacral ligaments (rotationally unstable, vertically stable)
            - APC III: hemipelvis separation with complete disruption of pubic symphysis and posterior ligament complexes (completely unstable)
    - lateral compression (LC)
        - typically result from side on impacts
        - common feature is a transverse fracture of the pubic rami
        - LC I: posterior compression of sacroiliac joint without ligament disruption (stable)
        - LC II: posterior SI ligament rupture, sacral crush injury or iliac wing fracture (rotationally unstable, vertically stable)
        - LC III: LC II with open book (APC) injury to contralateral pelvis (completely unstable)
    - vertical shear injuries (VS)
        - typically result from falls from height or head on motor vehicle crashes
        - common feature is a vertical fracture of the pubic rami
        - displaced fractures of the anterior rami and posterior columns, including SI dislocation (completely unstable)

- combined mechanism (CM) fractures
    - massive pelvic injuries that do not fit the other categories (completely unstable)

- haemorrhage from pelvic fractures
    - four potential sources:
        - surfaces of fractured bones
        - pelvic venous plexus
        - pelvic arterial injury
        - extra-pelvic sources (present in 30% of pelvic fractures)
    - classically venous haemorrhage is said to account for 90%, arterial for 10%
        - arterial bleeding is more common than this in patients with ongoing bleeding (e.g. despite pelvic binding or mechanical stabilisation) or have haemodynamic compromise
    - it is essentially bleeding into a free space that can accommodate the patient's entire blood volume without tamponade
        - true pelvic volume is around 1.5 litres but this is increased with disruption of the pelvic ring
        - the tamponade effect of the pelvic ring is lost in severe pelvic fractures with disruption of the parapelvic fascia
        - pelvic fractures cause bleeding into the retroperitoneal space
            - even when intact the retroperitoneal space can accumulate 5 litres of fluid with a pressure rise of only 30mmHg
        - haemorrhage can escape into the peritoneum and thighs with disruption of the pelvic floor (e.g. open book fractures)
    - haemodynamically unstable open pelvic fractures have mortality rates as high as 70%
- clinical assessment
    - should be as part of a structured ATLS approach
    - assessment of the pelvis should be performed with extreme care
    - inspect
        - ecchymosis
        - deformity
        - asymmetry
        - wounds
    - palpate
        - palpation of the skeletal structures:
            - pubic symphysis
            - iliac crests
            - posterior sacroiliac joints
            - ischial tuberosities
            - spine extending inferiorly to the sacrum and coccyx
    - assess for mobility
        - gently compress the iliac crests to feel for instability
        - if no pain or movement on compression, consider gentle distraction of the iliac crests
            - some experts advice against this

        - a gentle and cautious approach is required to avoid aggravating haemorrhage if the pelvis is fractured
        - the manoeuvre should only be performed once, ideally by the most senior trauma doctor present
        - do not 'rock' the pelvis
    - additional examination in patients with suspected pelvic fracture:
        - rectum
            - digital rectal examination to palpate for rectal injury (e.g. blood, wounds), bony fragments, sphincter function, boggy or high-riding prostate
        - perineum and genitalia
            - coexistant genital trauma, blood at the meatus, scrotal or other perineal haematomas
            - vaginal examination in women for vaginal tears
        - lower limbs
            - length discrepancy and malrotation, neurology
        - abdomen
            - tenderness, distension, external signs of trauma
    - normal examination in an alert adult patient effectively rules out significant pelvic injury (93-100% sensitivity) unless there are distracting injuries
        - any injuries missed in this circumstance tend to be clinically insignificant or require only conservative management
- investigations
    - bedside
        - VBG
            - monitor haemoglobin, lactate and acidaemia in major haemorrhage
        - FAST scan
            - assess for intraperitoneal fluid in a haemodynamically unstable patient with suspected pelvic fracture
            - positive scan suggests haemorrhage from intra-abdominal injury and the need for laparotomy
            - false positives may result from associated bladder rupture
        - diagnostic peritoneal lavage
            - can be used to rule out a false positive FAST scan in a haemodynamically unstable patient
            - performed above the umbilicus in patients with suspected pelvic fracture to avoid aspirating a pelvic haematoma
            - a positive result is aspiration of 10ml frank blood or GI contents
            - open procedure performed by a surgeon skilled in the technique, may take 20 minutes
    - laboratory tests
        - G&S, or cross-match 4-8 units if severely injured
        - FBC and coagulation profile
        - pregnancy test in women of childbearing age
    - imaging
        - AP pelvis x-ray

- normal x-ray does not exclude pelvic fractures completely, but does rule out pelvic fracture as a cause of haemodynamic instability
- indications for pelvic x-ray include:
  - haemodynamically unstable
  - altered mental state
  - distracting injuries
  - children (physical exam is less reliable)
  - abdominopelvic CT not being done for another reason
- do not perform if:
  - normal examination and the patient is alert and able to ambulate
  - abdominopelvic CT will be performed for another reason

▪ CT abdomen and pelvis with contrast
- imaging modality of choice for assessing pelvic ring injury
- performed in the haemodynamically stable patient to rule out intra-abdominal and retroperitoneal injury and to characterise the type and severity of pelvic injury; may identify those suited to interventional radiology

▪ angiography
- used to identify arterial injury and guide embolization

- associated injuries
  - abdominal and gastrointestinal injuries
    - rectal injury is common (up to 5%), other intestinal injury may also occur (up to 5%)
      - signifies an open fracture, which are more likely to be haemodynamically unstable
      - may require faecal diversion, pre-sacral drainage and perineal debridement
      - risk of death from secondary sepsis
      - may also have injury to spleen and liver (12%)
    - genitourinary injuries
      - bladder and urethral injury (5-20%)
      - urethral
        - posterior urethra with pelvic fractures
        - anterior urethra with straddle injuries
      - bladder
        - intra- and/or extra-peritoneal
      - vaginal tears
        - <5% in females, signifies an open fracture
  - other injuries (e.g. head, chest) may also be present, especially as the presence of a pelvic fracture implies a high energy mechanism of injury
- complications
  - acute
    - major haemorrhage and shock (leading mechanism of death)
      - laceration of venous structures
      - arterial injury (e.g. branches of internal iliac)
    - visceral and soft tissue injury

                - fractures may be compound into the perineum or vagina
                - lacerations into the rectum or bladder
                - urethral injuries common in males
            - nerve injury
                - sacral plexus injury (e.g. S2-5 sacral nerve root injuries with sacral fractures)
                - injuries to L4/5 or S1 nerve roots
            - ileus
            - fat embolization
            - acute respiratory distress syndrome
            - venous thromboembolism
            - abdominal compartment syndrome
        - late
            - infection (second most common mechanism of death)
            - fracture complications (e.g. osteoarthritis, malunion)
            - disability and immobility
            - incontinence
            - sexual dysfunction
            - dystocia in subsequent pregnancy
- management
    - resuscitation
        - co-ordinated team -based ATLS approach to address immediate life threats and identify other potentially serious injuries
        - commence haemostatic resuscitation if appropriate
        - pelvic stabilisation
            - avoid unnecessary movement of patient
            - apply pelvic binder early
            - if intubation required, pelvic binder should be applied first as neuromuscular blockade may allow pelvic volume to expand
    - specific initial management if haemodynamically stable
        - apply pelvic binder
        - abdominopelvic CT with IV contrast +/- CT cystography to identify abdominal and pelvic injuries and allow prioritisation of management
        - a pelvic 'blush' indicates the need for angiography and selective embolization of the actively bleeding artery
        - non-emergent surgical fixation as required
    - specific initial management if haemodynamically unstable
        - commence haemostatic resuscitation
        - apply pelvic binder
        - perform bedside tests:
            - AP pelvis x-ray
                - if normal, rules out pelvic fracture as cause of haemodynamic instability
            - eFAST
                - check for haemo/pneumothorax and intra-abdominal free fluid
                - if positive eFAST (or negative eFAST and positive DPL)

                - emergency laparotomy required during which pelvic stabilisation +/- pre-peritoneal pelvic packing is performed pending definitive management of the pelvic injury
            - if eFAST negative, manage as haemodynamically unstable patient with isolated pelvic trauma
        - CT with contrast once stabilised
- supportive care and monitoring
    - including neurovascular observation
- seek and treat complications
- disposition
    - depends on investigations, may include:
        - operating theatre
        - interventional radiology suite
        - intensive care
- management of the haemodynamically unstable patient with isolated pelvic fracture
    - uncommon
        - there are usually associated injuries due to the high energy mechanism of injury
        - approach is controversial and varies between centres according to available resources and protocols
            - the main goal is to stop the bleeding
    - approach
        - commence haemostatic resuscitation
        - apply pelvic binder
        - call trauma surgeon, orthopaedic surgeon and interventional radiologist on call
        - three management options can be performed in combination and in different orders:
            - angiography with embolization
                - may be immediately available in some centres
                - treats arterial bleeding, which is less common than venous bleeding but occurs more frequently in persistently hypotensive patients
                - either selective embolisation or non-selective embolisation can be done
            - packing
                - primarily stems venous bleeding, but the patient may be transferred for angiography post-packing
                - preperitoneal or direct retroperitoneal packing may be performed
            - mechanical stabilisation by external fixation
                - may be performed in the angiography suite or theatres
                - helps reduce bleeding from the venous plexus and from cancellous bone

        - does not offer any advantages over pelvic binding in the first instance, although pelvic binders may impair surgical access

TP7 limb and joint injury

- overall approach to major trauma involving a limb injury
    - resuscitation
        - ABCDE approach with cervical spine immobilisation if indicated
    - address life threats
        - pelvic fracture with major haemorrhage
            - pelvic binder
            - haemostatic resuscitation
            - correct coagulopathy
        - major arterial haemorrhage
            - direct pressure
            - tourniquet
            - elevate
            - haemostatic resuscitation
            - correct coagulopathy
        - crush syndrome
            - fluid resuscitation to keep urine output >1-2ml/kg/h
            - treat hyperkalaemia
    - address limb threats
        - open fractures
            - clean and cover wounds
            - reduce fracture
            - splint and elevate limb
            - antibiotics
        - compartment syndrome
            - assess compartment pressures and neurovascular status
            - remove constrictions
            - arrange for fasciectomy
        - amputation
            - preserve amputated part (clean, wrap in saline soaked gauze, keep on ice)
            - clean and cover wound
            - antibiotics
            - consider reimplantation
        - vascular injury
            - assess for hard and soft signs
            - measure ABI
            - consider CT angiogram and surgical intervention
        - neurological injury
            - assess neurovascular status
            - reduce fractures and relieve constrictions
            - consider surgical repair

        - degloving injury
            - clean and cover wounds
            - antibiotics
    - supportive care and monitoring
        - early analgesia
        - antibiotics (in severe open injuries)
        - tetanus prophylaxis if indicated
        - splint and elevate injured extremity
        - seek and treat other injuries (e.g. tendon rupture)
        - seek and treat complications (e.g. compartment syndrome, neurovascular compromise)
    - disposition
        - urgent surgical review for assessment, admission and operative intervention
        - transfer to specialist trauma centre if indicated
- potentially life-threating extremity injuries
    - pelvic disruption with massive haemorrhage
    - severe arterial haemorrhage
    - crush syndrome
- potentially limb-threatening injuries
    - open fractures/dislocations
    - traumatic amputation and severe vascular injuries
    - compartment syndrome
    - neurological compromise due to limb injury
    - degloving injuries
- crush syndrome
    - background
        - complex of electrolyte disturbances, metabolic acidosis and rhabdomyolysis resulting from crush injury
    - recognition
        - suspect based on history of crush injury or entrapment
        - hyperkalaemia, hypocalcaemia, hyperphosphataemia and hyperuricaemia from cellular damage
        - lactic acidosis from hypoperfusion
        - elevated CK and myoglobinuria due to massive muscle damage
            - urine dipstick positive for blood but no red cells seen on microscopy
        - acute renal failure due to rhabdomyolysis
        - x-rays to assess for associated fractures
        - assess for compartment syndrome
        - assess for neurological compromise (weakness, paraesthesiae, loss of sensation, neuropathic pain)
        - assess for vascular compromise (hard and soft signs of vascular injury, ankle brachial index, CT arteriography)
    - management
        - resuscitation of shocked patient
        - IV hydration (e.g. Hartmann's) to target urine output of 1-2ml/kg/h
            - corrects hypoperfusion, lactic acidosis, ameliorates AKI
        - aggressively treat potentially life-threatening hyperkalaemia

                - calcium gluconate
                - salbutamol
                - insulin
                - haemodialysis
            - calcium administration may lead to metastatic calcification in the presence of hyperphosphataemia
            - treat associated injuries including fracture/dislocations, wounds, neurovascular injuries, compartment syndrome
            - early analgesia, antibiotics if needed, tetanus immunisation/IgG
            - no good evidence for urinary alkalinisation with sodium bicarbonate or for mannitol
- traumatic amputation
    - recognition
        - usually obvious
        - check for associated neurovascular complications and crush injuries
        - bleeding may be slight due to arterial spasm
        - severed nerves are exquisitely painful
        - determine time of injury
            - reimplantation is less likely to be successful if warm ischaemia time exceeds 6 hours (in general) – but success has been achieved at up to 24 hours
        - x-ray the amputated part and the stump to help determine extent of injury and viability
    - management
        - consult surgery (may require general surgeon, plastics and/or orthopaedics)
        - always treat an amputated part as if it may be reimplanted
            - may at least be useful for achieving skin coverage of a wound
        - handle the amputated part with care
            - do not debride it
            - irrigate with normal saline
            - pack loosely with saline soaked sterile gauze
            - place in a water tight plastic bag and store in an ice water slurry
            - ensure ice does not directly come into contact with the amputated part
        - irrigate the stump with saline and control bleeding with direct pressure
        - prophylactic IV antibiotics, analgesia, tetanus prophylaxis
        - reimplantation is more likely to be performed for:
            - short ischaemia time
                - 1 hour of warm ischaemia equals 6 hours of cold ischaemia
            - thumb and index fingers are usually reimplanted
            - children
            - multiple amputations
            - dominant limb involved
            - patient's occupation depends on motor skills
            - upper limb amputations are more likely to be reimplanted than lower limb amputations as effective prostheses are more available for the latter and they are more likely to have crush injuries

        - a major trauma patient requiring resuscitation and emergency surgery is generally not a candidate for reimplantation
- neurological compromise due to limb injury
    - recognition
        - suspect nerve injury if vascular injury is present, as nerves tend to run in close proximity
        - detailed motor and sensory examination distal to the injury site
            - e.g. loss of function, weakness, areflexia, paraesthesiae, sensory loss
        - consider coexistant vascular injury, compartment syndrome and associated fracture
    - management
        - orthopaedics review (or hand surgeon/plastic surgeon as appropriate)
        - treat compartment syndrome if present
        - reduce and splint fractures
        - elevate limb to decrease oedema
        - rest affected limb in position of function
        - most closed soft tissue injuries with neurological injury gradually resolve over 3 months
        - transected nerves require operative repair, usually within 24 hours
            - unless minor sensory alterations only, which may be followed up at one week
- degloving injury
    - background
        - separation of the skin and underlying subcutaneous connective tissue from the underlying fascia
        - usually, but not always, open injuries causing exposure of the underlying structures
        - associated with high morbidity
    - recognition
        - usually easily identifiable by exposure of the underlying fascia investing muscles, vessels, nerves and bone
        - closed degloving injuries may not be obvious and are often missed on initial assessment
            - suspect based on mechanism that involves shearing forces and subcutaneous swelling suggesting underlying haematoma and tissue injury (e.g. run over by motor vehicle, limb caught in heavy machinery)
        - assess distal perfusion and neurological function
        - x-rays to look for fractures and foreign bodies
        - ultrasound may show underlying haematoma, soft tissue disruption, foreign bodies
    - management
        - urgent consultation with orthopaedic or plastic surgeon
        - clean and cover wounds with saline-soaked dressings
        - analgesia, antibiotics, tetanus prophylaxis
        - splint and elevate limb
        - preserve amputated parts

        - treat associated injuries and complications (e.g. fractures, dislocations, compartment syndrome, crush syndrome)
        - surgical treatment aims to achieve coverage by replacing the degloved tissue or through use of flaps or skin grafts to prevent necrosis of underlying structures
        - closed degloving injuries may be treated by washout and drainage of the subcutaneous tissues followed by compression bandages if the overlying tissues are viable
- open joint injury
    - background
        - also known as traumatic arthrotomy
        - soft tissue injury that penetrates the joint space and exposes the joint space to the environment
        - increases the risk of joint infection and warrants emergent orthopaedic evaluation with joint exploration and washout
        - rarely life-threatening
            - evaluate other injuries that may cause immediate loss of life or limb first
            - evaluate nearby neurovascular structures
        - maintain high suspicion for periarticular penetrating injuries to involve the joint
            - septic arthritis is a potential consequence
    - clinical features
        - concern for joint space involvement with soft tissue injury
            - proximity of soft tissue injury to joint
            - visible joint capsule surface
            - periarticular fracture
        - situations where further investigation is unnecessary and surgery/washout indicated:
            - foreign body in joint on x-ray
            - intra-articular air on x-ray or CT
            - obvious joint involvement of fracture with an open fracture on x-ray
    - investigation
        - ATLS approach
        - x-ray
        - CT – look for air in joint
        - trauma bloods if indicated
    - management
        - analgesia
        - wound management
            - initial immobilisation as indicated
            - surgical debridement and washout should occur within 6 hours
            - grossly contaminated wounds can be irrigated in the ED
            - tetanus prophylaxis as indicated
        - prophylactic antibiotics as per local guidelines for open fractures

TP8 burns

- background
    - in the UK 130,000 people per year attend the ED with burn injuries
        - around 8% are admitted
        - there are around 200 deaths per year
    - flame injuries are the most common cause of burns, followed by scalds
        - in children scalds are the most common
- definition and types of burn
    - definition
        - a traumatic injury to the skin or other organic tissue primarily caused by thermal or other acute exposures
    - types
        - thermal
            - most common type of burn
            - includes flame burns, scalds and contact burns
        - chemical
            - acids and alkalis can cause very deep burns through coagulative and liquefactive necrosis
            - they will continue to burn the skin until completely removed
                - it is essential that the skin is thoroughly irrigated
            - alkalis penetrate deeper than acids and require immediate attention
        - electrical
            - electrical current creates an entry and exit point, damaging tissue along its path as it is converted from electrical to thermal energy
            - electrical burns from domestic low voltage exposures tend to be less severe than high voltage burns which can cause extensive tissue damage and limb loss
            - domestic electrical burns should still be taken seriously and an ECG must be performed as alternating current can cause arrhythmias
        - cold exposure (frostbite)
            - burns are caused by ice crystals which can form both intra- and extra-cellularly
            - the subsequent fluid and electrical fluxes cause cell membrane lysis and cell death and a damaging inflammatory process is set up
        - radiation
            - radio frequency energy or ionising radiation cause tissue damage
            - the most common type of radiation burn is sunburn
            - other patients at risk are those undergoing radiation therapy for cancer treatment
- high risk groups in burns
    - children
        - infants and toddlers up to 4 years make up 20% of burn injuries
        - 70% are scalds due to spilling hot liquids or being exposed to hot bath water
        - non accidental injury must be actively excluded
    - older adults
        - people over 65 make up around 10% of patients with burns

        - may be due to slower reactions, mental and physical comorbidities and immobility
        - higher incidence of burns in winter months
        - non accidental injury must be actively excluded
    - other high risk groups
        - alcoholics
        - epileptics
        - chronic psychiatric or medical conditions
        - low socio-economic status
- pathophysiology
    - local response
        - zone of coagulation
            - occurs at the point of maximum damage (nearest point to the heat source)
            - there is irreversible tissue necrosis due to coagulation of proteins
        - zone of stasis
            - surrounds the zone of coagulation
            - characterised by decreased tissue perfusion
            - it is damaged but potentially viable
            - if the burn is managed correctly it has the potential to be salvaged, otherwise it could evolve into an area of necrosis
        - zone of hyperaemia
            - the outermost zone, where there is increased tissue perfusion
            - it is a reversible zone
    - systemic response
        - the release of cytokines and other inflammatory mediators at the site of injury has a systemic effect once the burn reaches 20-30% of total body surface area
        - cardiovascular changes
            - capillary permeability is increased, leading to loss of intravascular proteins and fluid into the interstitial compartment
            - peripheral and splanchnic vasoconstriction occurs
            - myocardial contractility is decreased
            - these changes, coupled with fluid loss from the wound, result in systemic hypotension and end-organ hypoperfusion
        - respiratory changes
            - inflammatory mediators cause bronchoconstriction
            - in severe burns, adult respiratory distress syndrome can occur
        - metabolic changes
            - the basal metabolic rate increases up to three times its original rate
            - coupled with splanchnic hypoperfusion this necessitates early and aggressive enteral feeding to decrease catabolism and maintain gut integrity
        - immunological changes
            - non-specific down-regulation of the immune response affecting both cell mediated and humoral pathways
- initial assessment and resuscitation

- primary survey with ABCDE approach to ensure that any life-threatening issues are not missed
- airway
    - at risk by three major mechanisms:
        - generalised oedema as a systemic response from an increasing burn size and depth can cause airway oedema and compromise airflow
        - localised oedema as a result of direct thermal damage to the airway can obstruct airflow
        - inhalation injury as a result of heat, smoke or toxic chemicals can cause damage to the airway
    - assessment
        - airway compromise may develop over a couple of hours and may only come to light when the patient is in crisis
        - factors increasing suspicion of airway obstruction or inhalation injury include:
            - hoarse voice
            - respiratory distress/stridor
            - carbonaceous sputum
            - singed nasal/facial hairs
            - inflamed oropharynx
            - burns to the face/oropharynx
            - history of burns in an enclosed space
            - raised blood gas carbon monoxide level
    - management
        - sit patient upright
        - senior anaesthetic review to identify and predict deterioration
        - if indicated, early intubation with an uncut tube prevents the tube from moving out in the event of further swelling
    - it is vital to predict and recognise the deteriorating airway
- breathing
    - gas exchange can be compromised for a number of reasons:
        - direct damage from inhalational injury to lower airways and gas exchange surfaces
        - carbon monoxide can quickly build up, impairing oxygen carrying capacity
        - burnt tissues with significant loss of elasticity in superficial fibres (eschar)
            - creates a constricting effect and inhibits expansion
            - when circumferential around the torso/chest/neck it can lead to ventilation issues
    - assessment
        - exposure of the chest to assess for any injuries and adequacy of ventilation
        - prompt assessment of oxygenation with a saturation probe
        - baseline blood gas to assess oxygenation, ventilation and carbon monoxide
    - management

- initially high flow oxygen
    - caution for falsely elevated peripheral saturation readings with raised carbon monoxide
    - titrate later to target appropriate saturations
    - immediate discussion with burns centre if any restriction of movement of chest
    - suspected inhalational injury may warrant intubation
- pitfalls and tips
    - it is important to recognise rising CO level
    - recognition of poor ventilation and need for escharotomy
        - can be a lifesaving procedure
    - cyanide poisoning is common in patients who have been exposed to inhalation of burnt household items
        - in profound hypoxia consider early administration of cyanokit

- circulation
    - burns >15% total body surface area in adults and >10% in children can cause profound circulatory shock
        - from both large tissue fluid losses and an inflammatory response
    - haemodynamic instability is rarely due to the burn alone and other causes should be sought
    - circumferential limb burns can compromise blood supply distally
    - assessment
        - thorough assessment of the extent of the burn
            - <15% TBSA in adults and 10% in children do not require immediate fluid resuscitation
        - capillary refill time, blood pressure and mucous membrane assessment are important indicators of hydration status but may be hard to measure due to the location of the burn
        - early catheterisation is rarely immediately helpful in the ED but is important as urine output is a reliable sign for poor perfusion and can be a guide to ongoing resuscitation
        - in a significant burn, increased metabolic demand can cause organ dysfunction
            - baseline bloods to consider are FBC, U&E, coagulation, LFTs, amylase, CRP and capillary blood glucose
                - also helps to identify other issues impacting on the patient
            - if the patient is likely to go to theatre, a G&S is warranted
        - in circumferential limb burns, blood supply to the extremities should be checked regularly
            - doppler ultrasound can be used if it is not possible clinically
    - management
        - immediate IV access and, if required, fluid resuscitation
        - blood tests
        - evaluation of any areas of circumferential burns in limbs with constant reassessment of perfusion

                - any deterioration in the circulation to a limb could indicate ischaemia or compartment syndrome and warrants immediate discussion with a burns centre
                    - may require escharotomy or fasciotomy
            - if IV or IO access is not possible through unburnt skin, access is mandated through burnt tissue
            - CK is a useful test to assess muscle breakdown
        - disability and exposure
            - appropriate exposure is fundamental
                - full exposure is required for thorough assessment of size and depth of burns and for a good secondary survey
                - patients with burns are physiologically vulnerable to getting cold so it is important to keep them warm and minimise fluid loss
            - management
                - maintain body temperature by active and passive warming
                    - balanced with the need to adequately expose the burn to assess, photograph and clean
                - clean the burn with normal saline and cover with strips of clingfilm
                    - do not use clingfilm on the face
                - consider imaging (e.g. x-ray, CT) based on secondary survey findings
            - be careful not to miss other injuries not associated with burns (e.g. traumatic head injury, long bone fractures)
            - ensure adequate analgesia
            - have all members of the team present simultaneously during exposure of an area to evaluate and photograph and avoid repeated unnecessary exposure and heat loss
- analgesia
    - should be given early as the burning process is exceptionally painful
    - non-pharmacological methods
        - cooling the burn under cool running water
        - covering with clingfilm
            - also helps to reduce heat loss and risk of infection
    - pharmacological methods
        - simple analgesia such as paracetamol and NSAIDs
        - stronger analgesia such as opiates and ketamine
- assessing extent and depth of burn
    - extent of burn
        - all areas of the body must be exposed to accurately estimate % TBSA affected
            - essential to:
                - decide whether the patient requires fluid resuscitation
                - calculate the fluid requirement during the initial resuscitation period if needed
        - common ways to assess % TBSA include:
            - Lund and Browder charts
            - using the patient's palm (including finger and thumb surfaces) as an estimate of 1% TBSA

- rule of 9s
    - head = 9%
    - chest = 18%
    - back = 18%
    - arms = 9% each
    - legs = 18% each
    - perineum = 1%
- Mersey Burns app
    - enables the user to highlight areas of burn tissue on a model to accurately estimate % TBSA affected

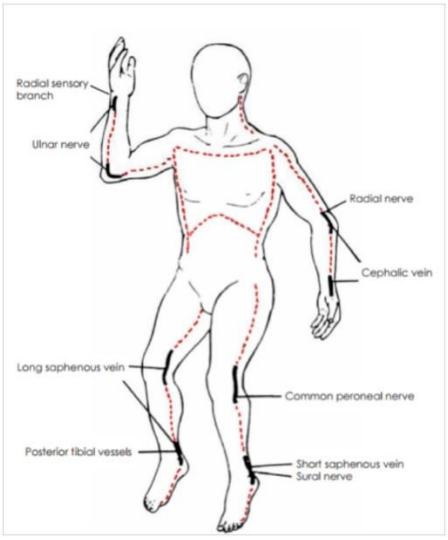
- not all of the burn area would necessarily be included in the calculation – the depth of burn needs to be considered

- depth of burn
    - to make an accurate assessment, skin needs to be cleaned, blisters removed (except for small non-tense blisters <6mm) and capillary refill time tested
    - the London and South East of England Burns Network has guidance on which blisters to deroof and how to do it
        - rationale for deroofing:
            - allows proper observation of the wound bed and accurate assessment of the burn depth, including capillary refill time and sensation, to determine appropriate treatment
            - removes non-viable tissue from the wound bed, allowing faster wound healing and decreasing the likelihood of scarring
            - evacuates blister fluid that may suppress local and systemic immune function, improving the patient's ability to defend against infection
            - reduces the risk of wound infection associated with uncontrolled blister rupture and prolonged presence of non-viable tissue
            - prevents pressure on underlying tissue, preserving the wound microcirculation and preventing the burn depth progression
            - enables movement of joints, reducing the likelihood of burn contracture
            - improves the efficacy of topical wound therapy
        - procedure for deroofing
            - should be done on the day of initial assessment to avoid re-adherence of non-viable tissue to the wound bed
            - administer analgesia and allow time to take effect – procedure may transiently increase pain
            - clean the wound with water or saline
            - remove all non-viable tissue from the wound bed using either mechanical debridement with moist gauze or sharp dissection with scissors and forceps
            - snip the blister, drain the fluid and cut away the dead or devitalised tissue carefully up to (but not including) the margin of sensate tissue

        - do not perform blister needle aspiration as bacteria may be introduced into the space and cause infection
        - send images of cleaned burn wounds to the local burn service
        - cover cleaned wounds with loose longitudinal strips of cling film for patients requiring prompt transfer to the local burns service
        - apply a non-adherent primary dressing with a secondary absorbent layer to optimise healing time, reduce hypertrophic scarring, improve the functional and aesthetic outcomes and offer a better option for comfort
        - do not use any topical agents unless advised by the local burn service after full deroofing
    - categorisation of depth
        - epidermal burn
            - affects epidermis only
            - skin is red and painful but not blistered
            - typical of sunburn
        - superficial partial thickness burn
            - affects the epidermis and upper layer of the dermis
            - the skin is pale pink and painful with blistering
            - capillary refill: blanches and rapidly returns
        - deep partial thickness burn
            - affects the epidermis and upper and deeper layers of the dermis
            - the skin appears dry or moist, blotchy and cherry red
            - may be painful or painless
            - there may be blisters
            - capillary refill: blanches with a sluggish return or does not blanch
        - full thickness burn
            - extends through all the layers of the skin to subcutaneous tissues
            - the skin is dry and white, brown or black in colour
            - no blisters
            - may be described as leathery or waxy
            - painless
            - capillary refill: does not blanch
    - burn wounds are often of mixed depth
    - epidermal burns are excluded from the % TBSA calculation when deciding on the need for resuscitation and fluid management
    - full thickness burns, depending on location, may pose a threat to ventilation or to limbs
- fluids
    - required for adults with >15% TBSA or children with >10% TBSA
    - the most frequently used formula is the Parkland formula
        - 2-4ml x weight in kg x %TBSA

        - estimates total resuscitation fluid required in first 24 hours from time of injury
        - is in addition to usual maintenance fluid requirement
        - 3ml is usually used for the calculation
            - 2ml in children or those at risk of fluid overload
            - 4ml in inhalational injuries where fluid losses likely to be greater
        - half of the calculated volume should be administered within the first 8 hours from time of burn
        - the remaining half should be administered over the subsequent 16 hours
        - optimal fluid choice is warmed balanced crystalloid such as Hartmann's or Plasmalyte
        - urine output should be closely monitored and input titrated to achieve an output of >0.5ml/kg/h in adults and >1ml/kg/h in children <30kg
        - urine output targets should be doubled if rhabdomyolysis is suspected
- management of the burn wound
    - remove
        - remove loose clothing and all jewellery
        - do not remove anything adherent or melted
    - cool
        - irrigate and cool thermal burns with cool (15 degree) running tap water for 20 minutes
            - can be beneficial up to 3 hours post injury
        - irrigate chemicals from skin/eyes immediately with warm running water for at least 15 minutes (longer irrigation periods usually required
        - do not use ice/iced water/ice packs/gel-based cooling products
    - cover
        - clean the wound with normal saline and cover any areas of skin loss with cling film
        - do not use cling film on the face
        - do not wrap clingfilm circumferentially around a limb
        - cover chemical burns with a non-adherent dressing
        - consider tetanus status
            - current guidelines suggest a burn would only be considered tetanus-prone in conjunction with signs of systemic sepsis
    - management of electrical burn wounds
        - maintain high level of suspicion for compartment syndrome
            - deeper tissue damage may be much more extensive than any superficial wounds may suggest and may involve whole compartments
        - tissue damage may lead to renal failure due to the release of haemochromogens into the circulation
            - if the urine is pigmented, output targets should be doubled
        - all patients should have an ECG – if the patient is asymptomatic and the ECG is normal, there is no indication for further cardiac monitoring or admission
- referral to specialist burns services
    - minimum threshold for referral (British Burn Association):
        - all burns ≥2% TBSA in children or ≥3% TBSA in adults

        - all full thickness burns
        - all circumferential burns
        - any burn not healed in 2 weeks
        - any burn with suspicion of non-accidental injury should be referred for expert assessment within 24 hours
    - factors prompting discussion with a consultant in a specialised burn care service with consideration of referral:
        - all burns to hands, feet, face, perineum, genitalia
        - any chemical, electrical or friction burn
        - any cold injury
        - any unwell/febrile child with a burn
        - any concerns regarding burn injuries and comorbidities that may affect treatment or healing of the burn
    - if the criteria are not met, continue with local care and dressings as required
    - if the burn wound changes in appearance or there are signs of infection or concerns regarding healing, discuss with a specialised burn service
    - if there are any concerns about toxic shock syndrome, refer early; consider with any size of burn if:
        - pyrexia
        - rash
        - diarrhoea/vomiting
        - general malaise
        - anorexia
        - tachycardia/tachypnoea/hypotension
        - reduced urine output
- escharotomy
    - background
        - may be required to allow adequate ventilation in circumferential chest burns or circulation in limb burns
        - the process of dermis stiffening takes time and patients often go to theatre for escharotomies at a later stage
    - procedure
        - keep the limb in the anatomical position
        - clean the area and incise along the anatomical lines with a scalpel down to fat



https://www.rcemlearning.co.uk/reference/major-trauma-burns/#1569851664441-abb39523-0813

- the incision should not go down to muscle or fascia
- for limbs the incisions need to release both medial and lateral aspects
- for the chest the incision needs to release the whole breast plate

TP9 inhalational injury

- background
  - injuries to the lungs and airway from smoke inhalation can be less apparent and may not present for 24-36 hours after exposure
- risk factors
  - being in a confined space
  - duration of exposure
  - substances being burnt that may emit poisons
  - pre-existing respiratory disease
- mechanism of injury
  - smoke inhalation injury leads to airway lumen narrowing or blockage through bronchospasm, increased mucus production, formation of airway casts, increased airway bloodflow
  - augmented airway bloodflow occurs immediately following inhalation injury and results in lung oedema and pulmonary transvascular fluid movements
  - obstructive casts from plasma exudates dimmish pulmonary function and may cause atelectasis, pneumonia and barotrauma respiratory distress
- clinical features
  - general
    - cough
    - wheeze
    - dyspnoea
    - irritated mucous membranes (runny eyes/nose)
    - chest pain
    - hypoxia
    - specific features dependent on type of exposure
  - inert gases

        - gases such as carbon dioxide and fuel gases displace air and oxygen, causing asphyxia
        - patients present with severe hypoxia
    - irritant gases
        - gases such as ammonia, formaldehyde, chlorine, nitrogen dioxide dissolve in water lining the respiratory tract and produce a chemical burn and inflammatory response
        - more soluble gases produce more upper airway burns/irritation symptoms
        - less soluble gases produce more pulmonary injury and respiratory distress
    - systemic toxins
        - includes carbon monoxide, hydrogen cyanide, hydrogen sulphide
        - interfere with the delivery of oxygen for use in cellular energy production
        - can cause liver, kidney, brain, lung and other organ damage
    - allergic
        - inhaled gases, particles and aerosols
        - produce bronchospasm and oedema similar to asthma
    - smoke inhalation/thermal
        - most fatalities from burn injuries are attributed to smoke inhalation
        - soot in posterior pharynx, singed nasal hair
        - hyperacute – severe wheezing, bronchoconstriction, significant hypoxaemia
        - acute pulmonary oedema – onset at 48-72 hours post injury in a previously asymptomatic patient
        - bronchopneumonia often at 10 days post injury
- types of inhalation
    - unintentional
        - smoke inhalation injury
            - cyanide
            - carbon monoxide
        - chloramine
        - hydrocarbons
        - sewer gas
            - hydrogen sulphide
            - ammonia toxicity
            - methane
            - carbon monoxide
    - terrorism
        - pulmonary chemical agents
            - ammonia
            - methyl isocyanate
            - methyl bromide
            - hydrochloric acid
            - chlorine
            - phosgene
        - bioterrorism
            - anthrax
            - smallpox
            - plague

                - tularaemia
                - botulinum toxin
                - viral haemorrhagic fever
                - ricin
        - burns
            - smoke inhalation injury
            - chemical injury
                - acrolein
                - hydrochloric acid
                - tuolene diisocyanate
                - nitrogen dioxide
            - systemic chemical injury
                - carbon monoxide toxicity
                - cyanide toxicity
            - specific types of burn
                - caustic burns
                    - airbag related
                    - tar burns
                    - cement burns
                - electrical injuries
                    - lightning injuries
- evaluation
    - evidence of exposure
        - estimated time of exposure
        - open or enclosed space
        - associated events such as fire, blast
        - whether the exposure is known
        - material on the patient
        - whether the patient smells of chemical
    - physical examination with focus on airway and pulmonary system
        - evidence of airway compromise or respiratory distress
    - CXR, pulse oximetry, ABG
    - consider carboxyhaemoglobin and/or cyanide level based on exposure history
- management
    - general
        - separate patient from fumes/toxic agents
        - decontaminate if not done on scene
        - secure airway if necessary and ventilate
            - otherwise observe for airway compromise and respiratory distress
        - oxygen at 6-12 litres/min via facemask
    - inert gases
        - remove victim from the gas
        - fresh air or oxygen
        - observe for sequelae from hypoxia (e.g. myocardial infarction, cerebral injury)
    - allergic
        - aerosolised bronchodilators

        - corticosteroids in patients a history of reactive airway disease
    - smoke inhalation/thermals
        - ensure adequate oxygenation, ventilation, pulmonary toilet and fluid resuscitation
- disposition
    - patients with respiratory distress or airway compromise require admission
    - observe for 1-4 hours otherwise
        - if no signs or symptoms of inhalational injury occur or if all resolved in 1 hour consider discharge home with instructions for return for re-evaluation next day or sooner if pulmonary and/or airway symptoms develop

TP10 wounds

- documentation of wounds
    - where and when the examination took place
    - accurate documentation of the type of injury
        - laceration, abrasion, incision etc.
        - does it match the alleged mechanism of injury
    - documentation of the shape of the injury, location and measurement from a fixed anatomical point
        - diagrams can be employed
    - documentation of colour
    - documentation of the presence or absence of active bleeding or clot
    - whether it appears to show signs of healing
    - documentation of what treatment, if any, was given
- abrasions

https://www.rcemlearning.co.uk/reference/soft-tissue-and-skin-injury-descriptions-in-the-emergency-department/#1573475297253-4728a055-bd03

    - grazes or scrapes
    - typically the result of tangential blunt force trauma exerting a dragging or frictional force to superficial skin
    - do not normally penetrate the full thickness of the epidermis
        - may do so focally
    - may bleed due to interruptions of the dermal papillae
    - end opposite point of impact generally shows heaped up epidermis
        - may have forensic significance in determining direction of force

    - varying types include
        - directional (brush type) resulting from contact with a rough surface (e.g. road rash)
        - fingernail scratches – often short and curved
    - assessment of age of abrasions can be difficult
        - may be helped by absence of bleeding, presence of scabbing, appearance of associated bruising
- bruises
    - extravascular collections of blood caused by various types of blunt force
    - may be patterned, can reproduce the shape of the weapon or object responsible
        - sometimes characteristic tramline bruise results from forceful contact with a rounded or squared-off weapon such as a baseball bat
    - bleeding can track under the skin
    - bruises are often larger where the skin is lax or there is underlying bone
        - in contrast to palms or soles of feet where skin is thick and bruises rarely seen
        - bruises may enlarge and track over time
    - assessment of age of bruise is not always reliable
        - colour changes do not occur in a predictable or linear fashion
        - most helpful colour is yellow, which does not appear in bruises less than about 18 hours old
- haematoma
    - palpable collections of blood, usually in muscle and soft tissue
    - often caused by direct blow
        - may have associated abrasion or laceration
    - scalp should be checked if there is the potential for blood to have tracked from elsewhere
- bites
    - pattern of injury produced by human or animal dentitions and associated structures
    - classified as a form of crush injury because the tooth compresses the skin, leaving an indentation or a break
    - usually has abraded and bruised components and often have a curved profile
    - can be a source of DNA
        - consider dry and wet swabs after discussion with the police if feasible and indicated before cleaning the wound
    - can be analysed by forensic odontologists
- lacerations
    - full thickness tears to the skin caused by blunt force trauma where tissues are crushed or torn apart by the object or weapon
    - often have the following features:
        - gaping
        - may be irregular, can also be linear
        - associated bruising (from being crushed)
        - associated abrasions to the edges
        - tissues bridges in depth of the wound
            - in contrast to incised wounds
        - rarely self-inflicted
        - presence of intact hairs which cross the wound

        - in contrast to incised wounds
    - relatively little blood loss (unless on scalp or intra-oral)
    - can be associated with fractures (e.g. underlying depressed skull fracture)
- incised wounds
    - follow sharp force trauma
    - may be divided into stab (puncture) wounds and cuts (slash wounds)
        - a stab wound is deeper than it is longer, a cut longer than it is deep
    - the shape can sometimes give an indication of the type of object used to cause the wound but may be difficult to determine in slash wounds
    - can be caused by anything sharp which impacts the body with sufficient force to penetrate the skin
    - wounds from heavy blades such as axes or machetes may have components of both lacerations and incised wounds
    - typical characteristics of incised wounds:
        - skin wound is often linear but can be jagged and irregular (e.g if caused by broken glass or bottles or if knife has moved in wound)
        - wound edges are cleanly divided
        - there is often no adjacent bruising of the skin edges
        - hair follicles are cleanly cut
        - the wound bleeds when the injury is sustained; bleeding can be heavy if vessels involved
- petechiae
    - documented presence or absence can be of forensic importance
    - may develop after alleged airway obstruction or from application of forceful neck pressure (strangulation)
        - needs to be documented and photographed at earliest opportunity as they disappear quickly
    - if an asphyxia component of an assault is reported, it is important to examine the face, especially around the eyes and eyelids (and after removal of any makeup)
    - petechiae can best be seen inside the eyelids and in the mouth
        - must be specifically looked for
    - other injuries relevant to the alleged assault (e.g. finger nail abrasions and fingertip bruising on the neck) must be carefully looked for
- investigations
    - obtain and record a verbatim report of how injuries sustained
    - used a ruler, scale or tape measure to accurately measure the size of injuries and the distance from a fixed structure
        - if guessing, the size should be recorded as approximate
    - photographs should ideally be taken if needed before treatment commenced
        - can be taken by police on body cameras, by Medical Photography or on a department camera
    - body maps can be used to document location of injuries
- management
    - varies depending on wound type and clinician preference
    - generally open wounds such as lacerations or incisions need to be cleaned and closed with steristrips, glue or sutures
    - consider tetanus status, particularly with deep contaminated wounds
    - wound may need to be explored and washed out if risk of foreign bodies

        - best done under local anaesthetic
    - x-ray may be indicated to look for foreign bodies or fractures
    - bites should be managed according to local protocol
        - usually involves thorough cleaning, sometimes with debridement, antibiotics, consideration of tetanus cover and BBV prophylaxis
    - never pass drains or lines through stab wounds or incorporate injuries into surgical incisions as the appearance of the injury is then distorted
- simple wound management and sutures
    - principles of wound management
        - assessment
        - haemostasis
        - analgesia
        - skin preparation and wound toilet
        - closure
        - dressing
        - infection prevention
        - follow-up
    - assessment
        - mode of injury
            - blunt
            - penetrating
            - blast
        - time of injury
        - type of wound
            - puncture
            - laceration
            - incision
            - crush
            - burst
            - bite
        - location
            - proximity to major vessels (potential damage to blood supply for healing)
            - nerves
            - organs
        - shape
            - linear
            - curved
            - stellate
            - Y-shaped
            - inverted V
            - etc.
        - depth and direction
            - risk to underlying tissues
            - skin tension lines
        - potential foreign body
            - suggestive history

        - whether it will be radio-opaque or require US location
    - potential underlying structural injury
        - bone fracture
        - tendon rupture
        - organ perforation
- haemostasis
    - spontaneous
    - management options
        - pressure
        - elevation
        - tourniquet
        - clamp/suture
- analgesia
    - local anaesthesia
        - topical (e.g. tetracaine/lidocaine combinations)
        - infilatrative
            - lidocaine
                - up to 3mg/kg
                - 1% solution contains 10mg/ml
        - caution advised for adrenaline in extremities (e.g. fingers, penis) but little evidence to support
- skin preparation and wound toilet
    - don't put alcohol or detergents inside the wound
    - tap water has the same or lower rates of infection as antiseptic solutions
        - sterile saline is usually used
            - 50-100ml saline under pressure (e.g. syringe with 25G needle)
    - irrigation
        - more important where there is high risk of infection
        - aim is to remove foreign matter and bacteria
    - consider debridement of ragged, non-viable skin edges
    - hair can be trimmed if necessary, but should not be shaved
    - remove foreign bodies but have personnel and equipment to control any bleeding
- closure
    - timing
        - primary closure
            - immediate closure for simple wounds <12 hours old (24h on the face) with opposable edges
        - delayed primary closure
            - if there is high risk of infection, prophylactic antibiotics and closure after around 4 days if no infection
        - secondary closure
            - allow the wound to close by itself:
                - bite (unless on the face)
                - separated edges
                - infection

        - may result in increased scarring
- options
    - steristrips
        - not for widely gaping or bleeding wounds
        - good for skin tears or to oppose bite wounds loosely, allowing for drainage
    - tissue adhesive
        - good for short lacerations with easily opposable edges
        - often used in paediatrics
        - a capillary tube, cannula tube of disposable pipette can help to apply the glue thinly
        - apply across opposed edges, not inside the wound
        - hold wound closed for around 30 seconds
        - forms an artificial scab and falls off in 7-10 days
    - sutures
        - type
            - absorbable (e.g. Vicryl) for deep sutures, or sometimes in children to avoid removal
            - non-absorbable (e.g. nylon, silk)
        - monofilament (less inflammatory response) vs braided (stronger knots)
        - needle: generally cutting edge rather than tapered end
    - staples
        - can be useful for scalp wounds
    - hair tie closure – note that hair trapped in the wound can impair closure
- techniques
    - generally interrupted sutures
        - mattress sutures may be required for larger wounds
    - first oppose the midpoint if linear or the corners if a jagged wound
    - ensure good bite of tissue taken with needle entering and leaving vertically
    - instrument tie with 3x double or triple throw knots
    - align knots outside of slightly everted laceration edges
    - space sutures 2-5mm apart
- suggested sizes and durations
    - child's face
        - 6'0 monofilament nylon
        - remove after 3-5 days
    - other parts of children
        - 5'0 catgut
        - deep part absorbs and the top part sloughs off after 10-14 days
    - adult's face
        - 5'0 monofilament nylon
        - remove after 5 days
    - adult hand
        - 4'0 nylon
        - remove after 7 days
    - adult scalp

        - 3’0 nylon/silk
        - remove after 5 days
    - adult arm/trunk/abdomen
        - 3’0 nylon/silk
        - remove after 9-14 days
    - adult leg
        - 3’0 nylon
        - remove after 14 days
- risk factors for delayed healing
    - size, location and motion of wound
    - age
    - genetics
    - race
    - Marfan’s syndrome, connective tissue disorders
    - nutritional deficiencies
        - supplements to non-deficient patients probably have little or no benefit
    - local infection
    - ischaemia
    - glucocorticoid therapy
    - diabetes mellitus
    - smoking
    - foreign bodies
- dressings
    - the first layer in contact with the wound surface should be non-adherent
        - e.g. lightly lubricated gauze with interstices
    - occlusive dressings can lead to maceration with retained fluid
    - the next layer should be absorbent material to attract any wound exudate
    - soft gauze and tape to secure in place
    - dressings may not be necessary if the wound is dry and extra protection is not required
- infection
    - signs and symptoms
        - increasing local inflammation
            - redness
            - pain
            - warmth
            - swelling
        - discharge/collection of pus
        - systemic signs – fever
    - risk factors
        - delayed presentation >12 hours
        - foreign bodies
        - heavily soiled wounds
        - bites
            - especially human, cats
        - puncture wounds

        - especially on the foot
    - intra-oral wounds
    - open fractures/exposed tendons
    - crush wounds
- antibiotic usage
    - advisable for high risk wounds or if there are signs of infection
    - lower threshold for hand and foot wounds
    - choice depends on most likely pathogen and local policy
        - human/cat/dog bites – co-amoxiclav
        - Staph/Strep spp – flucloxacillin/penicillin
        - Pseudomonas spp - ceftazidime

TC1 compartment syndrome

- background
    - increasing pressure in the fascial compartments of the limb leads to decreased venous outflow, which leads to further increase in intra-compartmental pressure, then compromised arterial inflow
        - this then leads to muscle ischaemia and necrosis
    - most common sites are:
        - lower leg
        - thigh
        - forearm
        - foot
        - hand
        - buttock
- causes
    - fractures
        - most common lower limb cause is tibial fracture
        - in the upper limb, injuries around the elbow and forearm
    - crush injury
    - burns
    - snake bite
    - prolonged surgical procedure
        - especially lithotomy position
- signs
    - symptoms tend to develop within hours
        - may present up to 48 hours after the insult
    - 6Ps
        - pain
        - paraesthesia (late)
        - paralysis (late)
        - pallor (late)
        - perishingly cold (late)
        - pulseless (very late)
    - pain is:
        - out of proportion with the history
        - worse on passive stretching of the affected muscles

        - worse on palpation of affected muscle groups
        - not improved with analgesia, elevation to the level of the heart, splitting a tight cast
- assessment
    - diagnosis mainly clinical
    - intra-compartmental pressure monitor if diagnostic uncertainty (e.g. unconscious/intubated)
        - if pressure >30mmHg or within 30mmHg of the diastolic pressure, compartment syndrome is likely
    - CK may be useful if rising
    - immediate fasciotomy or escharotomy required
- management
    - initial
        - keep limb at neutral level
        - high flow oxygen to improve oxygen delivery
        - IV fluids to improve blood pressure and transiently improve limb perfusion
        - removal of all dressings/splints/casts down to skin
        - opioid analgesia
    - fasciotomy
        - division of the fascia enclosing the involved muscular compartment via surgical incision
            - causes a release in pressure
        - incisions left open for 24-48 hours with relook to assess for dead tissue that may need debriding
        - in the lower leg, the anterior, lateral, superficial posterior and deep posterior fascial compartments must all be released
    - monitoring of renal function – possibility of rhabdomyolysis or reperfusion injury

TC2 limb and joint injury including bony, musculo-tendinous and complications

- bony
    - fractures
        - complications
            - general
                - haemorrhage and shock
                - thromboembolism
                - fat embolism
                - chest complications (e.g. chest infection)
                - urinary complications (e.g. UTI)
                - pressure sores
            - related to fracture
                - delayed union of fracture
                - malunion (deformity)
                - infection (especially with open fractures)
                - joint stiffness
                - compartment syndrome
                - neurovascular complications
                - avascular necrosis

        - particularly femoral head, humeral head, talus, scaphoid
    - related to treatment
        - damage to soft tissue
        - damage to neurovascular structures
        - damage to tendons and muscles
        - breakage of screws and rods
        - pin track infection
        - further surgical procedure to remove metal
- upper limb
    - wrist (carpal aspect)
        - scaphoid fracture
            - consists of proximal and distal segments separated by the waist
            - waist is most common location of fracture
            - may undergo avascular necrosis following fracture because blood supply is end artery running from distal to proximal (so proximal pole most prone to AVN)
            - usually caused by FOOSH
            - tenderness in anatomical snuffbox region
            - pain on telescoping thumb (causes metacarpal of thumb to hit injured scaphoid)
            - if fracture not seen on x-ray, repeat x-ray in 2 weeks
            - scaphoid cast for at least 6 weeks with hand in cup-holding position for confirmed fracture
        - lunate dislocation

https://radiopaedia.org/cases/74201/studies/85051?lang=gb&referrer=%2Farticles%2Flunate-dislocation%3Flang%3Dgb%23image_list_item_52024461

            - lunate dislocates in volar direction, can exert pressure on the median nerve leading to median nerve palsy
            - caused by FOOSH
            - swelling/ecchymosis at wrist +/- median nerve symptoms

- paraesthesia of radial 3 ½ fingers, weakness of abduction of pollicis brevis
- loss of normal appearance of lunate on x-ray (lunate normally looks like a cup holding and apple
- requires immediate reduction under analgesia/sedation +/- ORIF
  plaster immobilisation for 6 weeks

- perilunate dislocation

https://radiopaedia.org/cases/9893/studies/10459?lang=gb&referrer=%2Farticles%2Flunate-dislocation%3Flang%3Dgb%23image_list_item_470126

- very unstable but much less risk of median nerve injury than lunate dislocation
- history of FOOSH
- obvious deformity at the wrist with swelling +/- ecchymosis
- may have median nerve distribution paraesthesia if nerve has become stretched
- x-ray shows obvious dislocation of the carpus with the lunate in the normal position (apple out of cup on lateral but cup is upright)
- requires reduction under sedation + below elbow slab, may need K-wires +/- ORIF
- plaster for 6 weeks

- wrist (radio-ulnar aspect)
  - Colles' fracture

https://radiopaedia.org/cases/12382/studies/12645?lang=us&referrer=%2Farticles%2Fcolles-fracture%3Flang%3Dus%23image_list_item_612646

        - fracture of the distal inch of the radius with displacement and/or angulation of the distal fragment dorsally and radially
        - may also be impaction of the fracture fragments, shortening and associated fracture of the ulnar styloid
        - history of FOOSH
        - dinner fork deformity to distal radius +/- swelling and ecchymosis
        - x-ray shows fracture of distal radius with displacement and/or angulation of the distal fragment dorsally and radially
        - requires reduction under haematoma block or sedation using longitudinal traction, ulnar deviation, wrist flexion and counter-traction at the elbow
        - below elbow slab in wrist flexion and ulnar deviation
        - K-wiring +/- ORIF may be required if closed reduction unsatisfactory
        - plaster for 5-6 weeks
- forearm
    - mid-shaft radio-ulnar fracture
        - usually FOOSH
        - deformed forearm +/- ecchymosis
            - deformity may be slight if only one of the bones is fractured as the other splints
        - management:
            - closed fracture/no displacement
                - above elbow plaster
            - closed fracture/minimally displaced
                - MUA and above elbow plaster
            - closed fracture/grossly displaced
                - ORIF

- open fracture
    - Ex-Fix

- Monteggia fracture

https://radiopaedia.org/cases/10435/studies/10940?lang=us&referrer=%2Farticles%2Fmonteggia-fracture-dislocation%3Flang%3Dus%23image_list_item_535529

    - fracture of upper 1/3 of ulna with angulation and shortening with radial head dislocation (usually anterior)
    - direct blow to forearm or FOOSH
    - difficult to diagnose clinically, deformity and swelling present
    - true lateral views required to see radial head dislocation
    - requires ORIF with plating of fracture to restore original bone length
        - reduction of the dislocation usually occurs spontaneously after restoring bone length

- Galeazzi fracture

https://radiopaedia.org/cases/12221/studies/12534?lang=us&referrer=%2Farticles%2Fgaleazzi-fracture-dislocation%3Flang%3Dus%23image_list_item_590891

    - fracture of the lower 1/3 of radius with distal radio-ulnar joint dislocation

        - FOOSH, usually with elbow flexed
        - difficult to diagnose clinically, deformity and swelling present
        - requires ORIF with plating of fracture to restore original bone length
            - reduction of the dislocation usually occurs spontaneously after restoring bone length

- elbow

https://radiopaedia.org/cases/10355/studies/10875?lang=us&referrer=%2Farticles%2Fradial-head-dislocation%3Flang%3Dus%23image_list_item_530284

    - radial head fracture/dislocation
        - the annular ligament surrounds the radial head and is attached to the radial notch of the ulna, surrounding the radial head and neck like a pouch
        - history of FOOSH
        - swelling/ecchymosis at the elbow with pain on passive movement; no obvious deformity
        - true lateral x-ray is important
            - radial head not in alignment with capitellum (line drawn through the shaft of the radius – radiocapitellar line - should pass through both the radial head and the capitellum)
        - look for ulnar fracture, isolated dislocation is rare
        - fracture of the radial head may be:
            - undisplaced
            - displaced
            - comminuted
            - associated with radial neck fracture
        - dislocation – closed reduction with forced supination of forearm and direct pressure over radial head
            - above elbow plaster in supination for 2 weeks

        - undisplaced fracture - above elbow plaster in supination for 2 weeks
        - fragment or through-neck fracture - K-wire to radius and above elbow plaster in supination for 6 weeks, then remove wire
        - comminuted – excision of radial head +/- radial head prosthesis, above elbow plaster in supination for 6 weeks
    - supracondylar fracture

https://radiopaedia.org/cases/50191/studies/55538?lang=us&referrer=%2Farticles%2Fsupracondylar-fracture-summary%3Flang%3Dus%23image_list_item_27227452

        - most common fracture in children
        - history of FOOSH
        - swelling and ecchymosis at elbow
        - check pulses – distal supply may be compromised
        - check nerve supply – median, radial and ulna
        - severe fractures are usually obvious on x-ray as transverse fractures of the distal humerus above the condyles on lateral views
        - check radiocapitellar line passes through centre of capitellum on lateral view
        - look for fat pads on lateral view
            - anterior fat pad – triangular, ‘sail sign’, may be present normally
            - posterior fat pad – always abnormal
        - undisplaced, non-angulated fractures treated with above elbow plaster with elbow flexion for 6 weeks
        - if displaced or angulated may need K-wire fixation if closed reduction fails
        - check pulse/sensation after attempted reduction
    - elbow dislocation

https://radiopaedia.org/cases/14118/studies/13968?lang=us&referrer=%2Farticles%2Felbow-dislocation%3Flang%3Dus%23image_list_item_1167083

        - history of FOOSH with elbow in extension
        - obvious s-shaped deformity at the elbow
        - loss of the equilateral triangle formed by the medial and lateral condyles and the olecranon process
        - assess distal pulses and median nerve sensation
        - obvious dislocation on x-ray – look for associated fractures especially of the coronoid process of the ulna (may cause joint instability) or radial head
        - reduction of simple dislocation with traction on forearm and counter-traction on arm whilst pressure applied on the olecranon process
            - sling for 3 weeks then mobilise to prevent stiffness
- humerus
    - mid-shaft humeral fracture
        - history of FOOSH
        - ecchymosis of arm, may have deformity
        - x-ray shows spiral fracture usually, may be transverse or oblique, displaced or undisplaced
        - important to look for radiolucent areas suggestive of metastasis
        - undisplaced fractures managed with above elbow plaster for 3 weeks then changed to brace, with some angulation/displacement acceptable
        - if grossly displaced/angulated may require ORIF with plating
        - radial nerve injury is a possible complication
- shoulder
    - anterior shoulder dislocation

https://radiopaedia.org/articles/shoulder-dislocation?lang=us

- 90-95% are anterior
- anterior dislocations are usually caused by trauma, e.g. FOOSH
- posterior dislocations are usually caused by electrocution, epileptic fit or electroconvulsive therapy
    - can be caused by trauma
- inferior dislocations are classically caused by falling down a manhole with the arm impacting against the sides
- signs:
    - obvious deformity at shoulder joint – usually a flattening
    - adduction and internal rotation of the upper limb
    - acromion process is now most lateral point of shoulder
    - axillary fullness due to humeral head's new position
    - loss of sensation over deltoid area ('regimental patch') secondary to axillary nerve neuropraxia
    - assessment of distal pulses
- x-ray shows humeral head out of glenoid fossa and usually inferior to the coracoid process of the scapula
    - on lateral view the head is anterior to the Y formed by the scapula (gold ball anterior to the tee)
- look for associated fractures of humeral head and neck
- closed reduction under sedation or GA with:
    - steady traction of mildly abducted upper limb with whole body counter traction (variation of Hippocratic method)

- lying the patient prone with arm hanging over side of bed during traction (Stimson's method)
- Kocher's – flex elbow to 90°, laterally rotate arm 75°, abduct elbow after lifting forward, medially rotate arm
    - associated with increased risk of fracturing humeral neck

▪ body sling for 2-3 weeks then mobilisation with physiotherapy – may need open reduction if closed fails
▪ recurrent dislocation needs MRI to look for tear of glenoid labrum – requires operative stabilisation

- posterior shoulder dislocation

https://radiopaedia.org/cases/11813/studies/12176?lang=us&referrer=%2Farticles%2Fposterior-shoulder-dislocation%3Flang%3Dus%23image_list_item_577200

▪ arm held in fixed internal rotation, patient unwilling to move
▪ on x-ray, humeral head is usually in the glenoid fossa; humeral head has symmetrical shape on lateral view ('lightbulb sign') instead of the shape of a walking stick handle
▪ treatment is closed reduction with traction and lateral rotation of arm and full body counter-traction with direct forward pressure on the humeral head from behind
▪ sling for 2-3 weeks with mobilisation and physiotherapy afterwards

- surgical neck of humerus fracture
    ▪ anatomical neck is the articular margin of the humeral head, surgical neck is the upper end of the humeral shaft (more prone to fracture)
    ▪ history of FOOSH

        - arm may be in fixed internal rotation or angulated at the shoulder joint; ecchymosis
        - assess angulation, displacement and impaction of humeral shaft upwards into humeral head
        - impacted fractures are stable, managed with collar and cuff for 6 weeks followed by physiotherapy
            - whole humerus acts as one piece
        - angulated/displaced – humerus acts as two pieces, requires closed reduction with hanging slab to distal humerus and physio in 4-6 weeks
        - if humeral head in >2 pieces and shaft not impacted, ORIF required with plating or hemiarthroplasty
    - clavicular fracture
        - most commonly at middle third
        - history of FOOSH
        - usually deformity in clavicular region with tenting of the overlying skin; fractured end of proximal segment may pierce the skin
        - on x-ray, the proximal fragment is pulled upwards by the sternocleidomastoid, and the distal fragment downwards by the weight of the upper limb
        - conservative management in arm sling for 2 weeks then physio
        - deformity secondary to malunion is usually accepted
        - ORIF may be considered if:
            - grossly displaced fracture at risk of non-union
            - unacceptable cosmesis
            - open fracture
    - sternoclavicular dislocation
        - usually dislocates anteriorly
        - posterior dislocation is more dangerous due to potential damage to subclavian vessels
        - anterior dislocation usually caused by direct fall onto shoulder, sometimes by FOOSH
        - posterior dislocation caused by direct force to joint (e.g. RTA)
        - obvious deformity at SCJ – bulge if anterior, depression if posterior
            - assess distal pulses in posterior dislocation
        - diagnosis usually clinical, PA oblique or serendipity view shows nature of dislocation
        - treatment is non-operative, intervention only required if compromise to underlying vessels
        - sling and active physio after 2 weeks
- miscellaneous upper limb fractures

- Smith's fracture

https://radiopaedia.org/articles/smith-fracture?lang=us

    - fracture of distal end of radius with volar displacement (reverse Colles')
    - caused by fall onto back of wrist
    - reduction of fracture with K-wiring and above elbow plaster with wrist in slight extension for 6 weeks
- Barton fracture

https://radiopaedia.org/articles/barton-fracture?lang=us

    - fracture of distal end of radius which involves the radiocarpal joint (fracture line runs into joint)
    - distal fragment and entire carpus displaced in volar direction
    - unstable, requires ORIF and plaster for 6 weeks
- Bennett fracture

https://radiopaedia.org/articles/bennett-fracture?lang=us

- oblique fracture of base of 1st metacarpal extending into carpometacarpal joint
- distal fragment of metacarpal usually displaced
- attempt closed reduction and thumb spica but K-wiring may be needed

- metacarpal fractures
  - minimally or nondisplaced – neighbour strapping or below elbow plaster extending to over the involved fingers for 3 weeks with wrist and hand in safe position (wrist and hand 20° dorsiflexion, MCP joints at 90° flexion, interphalangeal joints in extension) – holds the ligaments taut so they do not contract and stiffen
  - needs K-wiring/ORIF if displaced or irreducible
  - assess for rotation of the fingers – all fingers should point towards thenar eminence or scaphoid when loose fist made
    - if there is rotation, closed reduction and neighbour strapping required
- Boxer's fracture

https://radiopaedia.org/articles/boxer-fracture-1?lang=us

        - fracture of distal end of little finger metacarpal
        - usually managed non-operatively with closed reduction and neighbour strapping for 2-3 weeks only (longer risks stiffness)
    - distal phalanx fractures
        - treated as soft tissue injuries with analgesia, elevation and ice
- lower limb
    - hip fracture
        - sources of blood supply to femoral head:
            - intramedullary arteries from the femoral shaft
            - retinacular arteries within the joint capsule, originating from the medial and lateral femoral circumflex arteries, running from distal to proximal artery of ligamentum teres
        - intracapsular fractures are within the hip joint capsule
            - risk of avascular necrosis due to disturbance of retinacular blood supply to femoral head
            - Garden's classification:
                - I: ends are impacted – best outcome as vascular regrowth is facilitated
                - II: non-impacted ends but minimal displacement of fragments
                - III: complete fracture with partial displacement of fragments
                - IV: complete fracture with total displacement
        - extracapsular fractures occur outside the capsule, usually between the greater and lesser trochanters
            - intertrochanteric most common
            - subtrochanteric less common and often pathological
        - history is usually of a fall
        - examination shows shortened, rotated lower limb and inability to weight bear
            - check for pulses and sensation distally
        - intracapsular fractures require excision of the femoral head and insertion of a metal prosthesis (hemiarthroplasty)
        - extracapsular fractures require ORIF of the femoral head to the neck using cannulated screws (usually dynamic hip screw) (head-preserving procedure)
    - hip dislocation
        - 90-95% are posterior
        - usually from RTAs, sometimes from falls or following hip replacements
        - may have sciatic nerve damage due to pressure from the dislocated head with foot drop
        - limb is shortened, adducted and internally rotated

    - knee should also be imaged
    - reduction under sedation/GA
        - stability assessment is performed at the same time
        - traction is applied (skeletal with pin through tibia, or skin)
        - reduction should be as soon as possible (delay, particularly >6 hours increases risk of AVN)
    - CT should be done to look for fractured acetabulum or unacceptable reduction
- femoral shaft fracture
    - can be midshaft or supracondylar
    - history of direct trauma, twisting injuries in sport, heavy falls; or pathological fractures
    - signs of shock, swollen and deformed limb
    - any open wound should be covered with a Betadine-soaked swab
    - analgesia and splinting with distal traction
    - ORIF if closed, Ex-Fix if open
    - for supracondylar fractures, above knee slab, and usually ORIF
- patellar fracture

https://radiopaedia.org/articles/patellar-fracture-2?lang=us

    - anatomy
        - quadriceps tendon attaches at the superior pole and patellar ligament at the inferior pole (also attaches to the tibial tuberosity)
    - history of direct fall onto knee with inability to straight leg raise
    - swelling and ecchymosis around knee, possible palpable crepitus
    - x-ray shows if fracture transverse (most common), longitudinal or comminuted

- transverse fractures managed with ORIF due to disruption of continuity of straight leg raising mechanism
- longitudinal/comminuted managed with long leg cylinder cast for 6-8 weeks

- patellar dislocation

https://radiopaedia.org/articles/lateral-patellar-dislocation?lang=us

- history of twisting injury of knee, more commonly in young girls
- deformed knee +/- swelling (depending on concurrent ligamentous injury), knee in mildly flexed position
- patella is usually laterally displaced
- x-ray confirms dislocation and rules out fracture
- management is reduction under sedation, applying pressure to the patella in the direction opposite to the dislocation whilst extending the knee

- tibial plateau fracture

https://radiopaedia.org/articles/tibial-plateau-fracture?lang=us

- involves the proximal end of the tibia and its articular surface
- the lateral tibial plateau is most commonly fractured
- history of RTA or fall (e.g. off horse, bike)

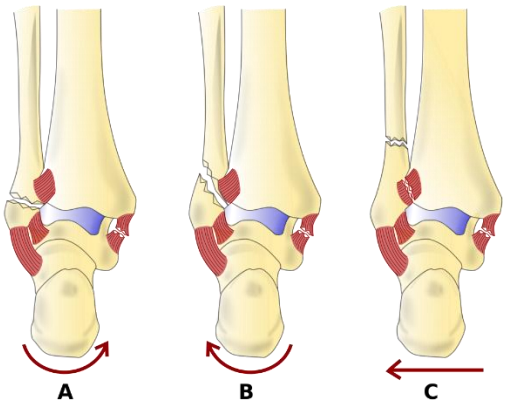
- pain, ecchymosis and swelling around knee; deformity unusual
- x-ray to assess whether plateau, condyle or both involved
- CT usually required to assess full extent
- management depends on the fracture; bone graft sometimes needed to strut fragments to normal positions and restore joint congruity
- plateau fractures require ORIF with plate and screws, usually with bone graft
- condyle fractures require ORIF with plate and screws +/- bone graft

- tibial-fibular fractures
  - high incidence of compound fractures requiring external fixation, wound infection and union problems
  - history is usually of a fall, sporting injury or RTC
    - isolated fibular fractures are usually secondary to a direct blow
  - assess distal neurovascular status
  - on x-ray check for displacement and angulation
  - if closed fracture, ORIF with intramedullary nail or plates/screws if displaced or angulated
    - if non-displaced, above knee cast for 8 weeks
    - if open, washout and debridement then Ex-Fix or IMN
  - union problems are more likely in isolated tibia fractures as the intact fibula acts as a strut keeping the ends apart
- ankle mortise fractures

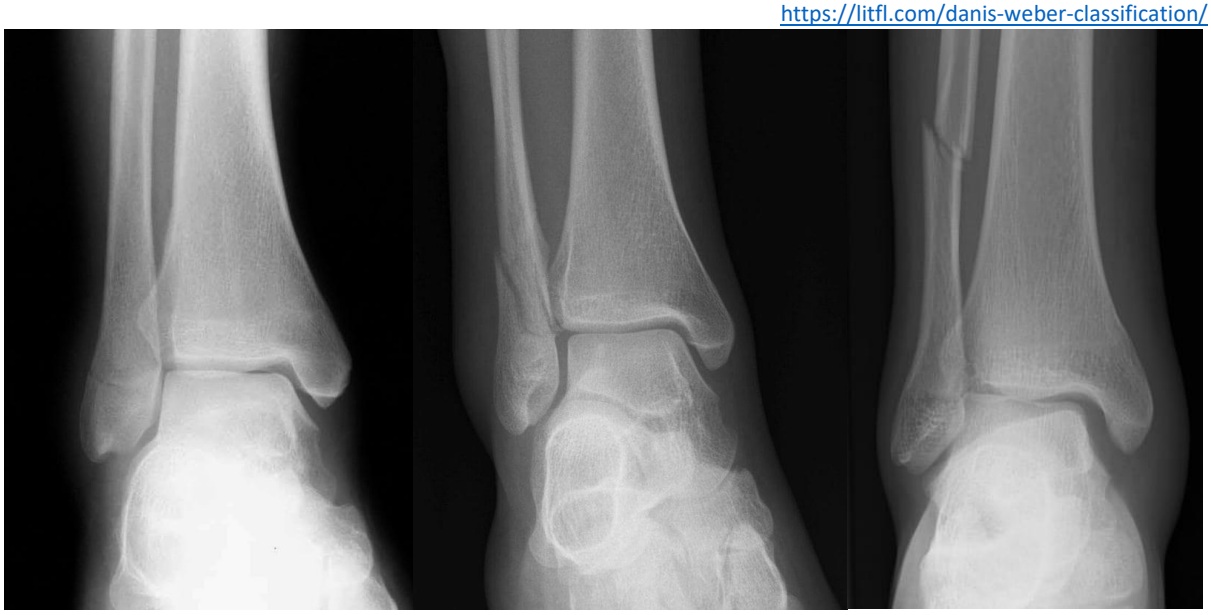
https://radiopaedia.org/articles/ankle-mortise-view?lang=us

  - the ankle mortise consists of:
    - distal articular surface of the tibia
    - medial malleolus
    - lateral malleolus

        - posterior malleolus (posterior part of the distal end of the tibia)
        - talus
    - the ankle mortise is stabilised by the distal tibiofibular syndesmosis:
        - anterior and posterior tibiofibular ligaments
        - inferior transverse ligament
        - interosseous ligament
    - bi-and tri-malleolar fractures are usually highly unstable and require ORIF
    - injury is usually twisting, often during sports
    - there is gross swelling and ecchymosis of the ankle with deformity
    - on x-ray, look for:
        - how many malleoli are fractured
        - avulsions which may represent severe ligamentous injury and be as unstable as full fractures
        - talar shift
            - even mild shift is a sign of instability
    - in isolated medial malleolar fracture, get knee views in case of neck of fibula fracture (Maisonneuve's fracture)
    - treatment depends on type of fracture – number of malleoli, degree of talar shift, displacement of fragments
        - most unstable ankles need ORIF
        - stable, undisplaced (usually unimalleolar) fractures need a below knee cast for 6-8 weeks
    - in fibular neck fractures, damage to the common peroneal nerve may result in lateral foot paraesthesia or foot drop
    - Weber classification:
        - Weber A – fracture of the fibula distal to the syndesmosis; oblique medial malleolus fracture may also be present
        - Weber B – fracture of the fibula at the level of the syndesmosis; may be stable or unstable based upon presence of deltoid ligament rupture or medial malleolus fracture
        - Weber C – fracture of the fibula proximal to the syndesmosis; unstable, usually associated with syndesmosis injury and may include medial malleolus fracture or deltoid ligament rupture



https://litfl.com/danis-weber-classification/



https://litfl.com/danis-weber-classification/

- talar fracture

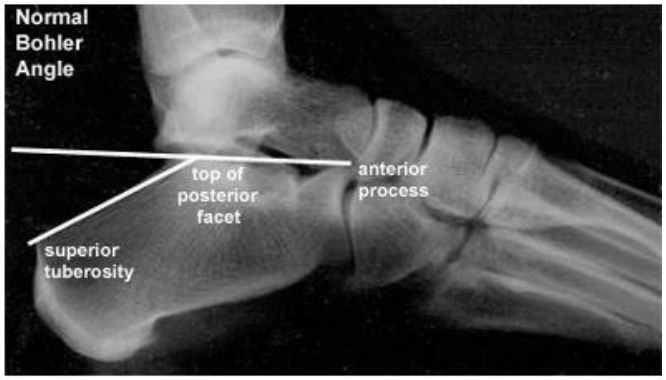
https://radiopaedia.org/articles/talar-fractures?lang=us

  - rare
  - secondary to high energy trauma
  - complicated due to complex anatomy and blood supply
  - managed with below knee cast if undisplaced, ORIF if displaced
    - may require CT scan

- calcaneal fracture

https://radiopaedia.org/articles/calcaneal-fracture?lang=us

  - caused by fall from a height
  - relatively common
  - has complex anatomy and blood supply
  - presents with severe swelling and pain of the heel/foot
  - examine back for vertebral crush fractures
  - requires plain film +/- CT of heel, and plain films of spine
  - the Böhler angle is measured on lateral views from the highest point of the anterior process to the highest point of posterior facet and a line tangential to the superior edge of the tuberosity
    - normally 20-40°
    - if less than 20° it represents a calcaneal fracture (collapse of the posterior facet)



https://www.orthobullets.com/trauma/1051/calcaneus-fractures

  - most are treated conservatively in below knee cast; some surgeons advocate ORIF (plate and screws)
- metatarsal fractures

        - common in inversion injuries of the foot
        - most commonly 5th metatarsal
        - managed with strapping, below knee cast or boot; ORIF rarely needed
    - phalangeal fractures
        - usually caused by stubbing the toe
        - treated conservatively with strapping of the toe
            - K-wiring occasionally needed, especially for the big toe

- musculo-tendinous
    - meniscal injury
        - medial and lateral menisci are crescent-shaped cartilaginous structures situated between the tibial plateau and the medial and lateral femoral condyles
        - medial meniscus is more commonly injured
            - twisting injury, usually sport related
        - presentation:
            - pain over the inner (or outer if lateral) aspect of the knee
            - swelling
            - locking (almost pathognomic)
                - caused by a torn bit of meniscus getting caught between the femoral condyle and tibial plateau during joint movement
            - giving way of the knee
        - examination:
            - swelling
            - may have locked knee
            - tender joint line on palpation either medially or laterally
            - positive McMurray's test
                - forced flexion and external rotation of the knee combined with medial compression of the joint line elicits pain (reversed for lateral meniscus)
        - investigated with MRI
        - treated with arthroscopic trimming of the tear and occasionally repair
    - ligamentous knee injury
        - medial and lateral collateral ligaments limit valgus and varus movement of the joint
        - anterior and posterior cruciates are within the joint, crossing each other, and limit anterior and posterior translation of the tibia
        - medial collateral is most commonly injured
            - usually due to valgus strain at the knee, commonly in sport
        - presents with pain and swelling +/- instability
            - there is pain on valgus strain
        - ACL rupture presents with pain, inability to weight bear and almost immediate swelling due to haemarthrosis
            - Lachmann test positive
                - patient lies down with knee flexed, tibia can be drawn forward in relation to the femur due to ACL laxity

        - anterior draw test is similar but with knee flexed to 90°
    - requires MRI of the knee and plain film to rule out bony injury
    - non-operative treatment if stable and low-demand patient, ACL reconstruction for unstable knee and high-demand patient
        - physio vital for rehabilitation
- Achilles' tendon rupture
    - the Achilles' tendon is the tendon of gastrocnemius, attached to the posterior aspect of the calcaneus
        - function is plantar flexion
    - ruptured by missing a step (especially during dancing), running or during sport (especially tennis)
        - steroids and use of fluoroquinolone antibiotics (especially ciprofloxacin) increase risk
    - patient typically notices a sharp 'snapping sensation' followed by an immediate inability to weight bear on that leg
    - clinical findings are an obvious gap in the tendon just above the heel which may be seen and felt (patient prone or kneeling on chair)
    - there is failure of the foot to plantar flex on squeezing the calf (Simmond's or Thompson's test)
    - non-operative management is above knee plaster with foot in equinus position (exaggerated plantar flexion) for 4-6 weeks, then can change to removable walking brace for further 4 weeks
    - operative is primary repair of the tendon and plaster in equinus for 4-6 weeks
        - outcomes are generally similar but risk of re-rupture is less with surgical repair
- ankle sprain
    - lateral ligament complex is most commonly injured
        - usually by inversion injury
    - presents with pain and swelling (less than in a fracture unless there is associated avulsion fracture)
        - there is no deformity but may be instability
    - categories of severity:
        - first degree (ligament injury without tear)
            - minimal function loss (patient ambulates with minimal pain)
            - minimal swelling
            - mildly tender over involved ligament
            - no abnormal motion or pain on stress testing
        - second degree (incomplete tear of ligament)
            - moderate function loss (patient has pain with weight bearing and ambulation)
            - moderate swelling, ecchymosis, tenderness
            - pain on normal motion
            - mild instability and moderate to severe pain on stress testing
        - third degree (complete tear of ligament)

        - significant function loss (patient unable to weight bear or ambulate)
        - egg-shaped swelling within 2 hours of injury
        - may be painless with complete rupture
        - positive stress test
- stress testing:
    - anterior draw test
        - flex knee, stabilise tibia by cupping the heel, move the ankle mortise joint in an anteroposterior direction
        - positive if there is asymmetric ankle excursion
    - talar tilt test
        - inversion at the ankle causing tilting and lifting of the mortise joint
        - positive if there is asymmetric ankle excursion
- x-ray if diagnosis uncertain
- Ottawa rules
    - ankle
        - pain in the malleolar zone plus any of:
            - bone tenderness at the posterior edge or tip of lateral malleolus
            - bone tenderness at posterior edge or tip of medial malleolus
            - inability to weight bear both immediately and in the ED
    - foot
        - pain in the midfoot zone and any of:
            - bone tenderness at base of 5th metacarpal
            - bone tenderness at the navicular
            - inability to weight bear both immediately and in the ED
- management
    - first degree sprain
        - rest, ice (every 2-3 hours for first 24 hours – causes local vasoconstriction, preventing further bleeding and slowing oxygen metabolism which reduces cell death and inflammatory response, also may reduce nerve conduction and provide analgesia), compression (most useful in first 3 days and avoided when limb is elevated; should be firm enough to assist in venous and lymphatic drainage but not compromise arterial flow), elevation
        - simple analgesia
        - tubi-grip or elastic support bandage
        - early weight bearing, aim for return to full activity within a week
        - follow up with GP or physiotherapist
- second degree sprain
    - rest, ice, compression, elevation

                - simple analgesia
                - ankle support – airsplint, ankle boot, bimalleolar orthotics (physiotherapist involvement recommended)
                - should not weight bear for first 48-72 hours – crutches to help with this
                - follow up with GP and physiotherapist
            - third degree sprain
                - rest, ice, compression, elevation
                - simple analgesia +/- further analgesia
                - management controversial – elastic bandage and early mobilisation or immobilisation in plaster cast or air splint for 10 days
                - close follow up with GP or orthopaedic surgeon and physiotherapist

TC3 electrical burns

- types of electrical injury
    - cardiac dysrhythmia
        - asystole (DC) and VF/VT (AC) are the most sinister rhythms
            - others can occur, e.g. atrial fibrillation, atrial tachycardia, junctional rhythms, SVT, first and second degree heart block, premature ventricular beats
        - ST segment abnormalities usually resolve spontaneously
        - coronary artery spasm and thrombosis can cause myocardial ischaemia and infarction
    - respiratory
        - apnoea can occur secondary to tetany of the respiratory muscles or disruption of the output of the respiratory centre in the brainstem
    - cutaneous burns
        - often full thickness involving entrance and exit sites such as hands and feet
        - may have a relatively small surface area and lead to underestimation of the amount of damage
    - peripheral neurology
        - immediate or delayed effects with paraesthesia and weakness
        - ulnar and median nerves are often involved with hand injuries
    - CNS features
        - electrical current to the brain can cause loss of consciousness, confusion, amnesia and seizures
        - findings such as hemiparesis, speech disturbance and visual disturbance can occur
        - involvement of the spinal cord can result in transient or permanent tetraplegia or paraplegia
            - incidence of spinal cord injuries after high voltage electrical accidents varies between 2 and 27%
    - musculoskeletal
        - deep muscle burns can cause contractures and deformity
        - muscle necrosis can cause electrolyte abnormalities, myoglobinuria and renal impairment

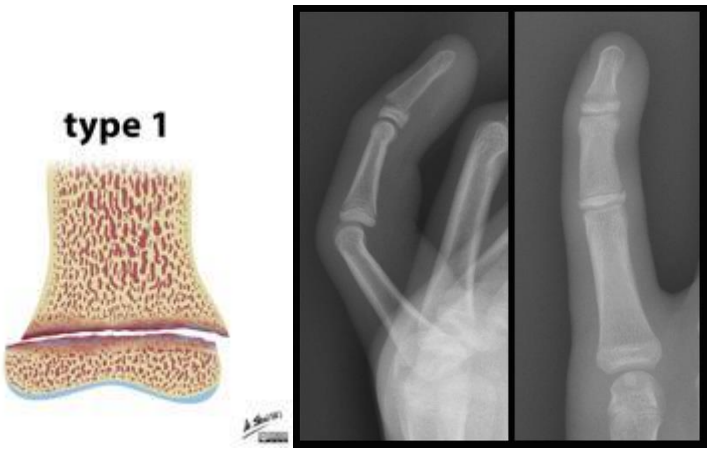
        - muscle oedema within myofascial sheaths can cause compartment syndrome
        - secondary trauma from being thrown, such as posterior shoulder dislocation
        - opisthotonos can cause vertebral fractures with damage to the cord or cauda equina
    - ocular
        - passage of current through or adjacent to the eye can cause burns to the cornea, sclera or deeper structures
        - cataract formation as a delayed consequence of lens involvement
    - vascular
        - vascular spasm and thrombosis with potential distal ischaemia
        - delayed aneurysm formation can occur from damage to the vessel wall
    - intramural burns
        - always check inside the mouth as it is an area of low resistance
        - intraoral burns are more common in young children – generally placing electrical cords in mouth
            - conduction can be aided by saliva
        - delayed oral haemorrhage can occur as vasospasm resolves
        - contraction of scar tissue can make intraoral burns disfiguring
    - effects on foetus
        - amniotic fluid conducts electrical current with potential devastating effects on the foetus if it is in the pathway of the current
        - maternal cardiac arrest and dysrhythmias also compromise uteroplacental blood flow
- investigations
    - ECG
        - probably useful for low voltage electrocution
        - patients with a normal ECG do not require further monitoring
            - risk of malignant dysrhythmia is greatest at the time and unlikely at a later stage
        - in high voltage injuries (>1000 volts), those who have loss of consciousness, transthoracic current pathway or abnormal baseline ECG probably require cardiac monitoring
            - evidence is lacking for need and duration of monitoring
    - troponin
        - consider baseline and 12 hour troponin if high risk features
        - clinical significance of elevation is uncertain
    - CTG
        - CTG monitoring of foetus for women over 22 weeks gestation
        - and obstetric consultation
- management
    - pre-hospital
        - switching off the current source
            - usually done by fire brigade or regional electrical authority
            - high voltage sources may cause arcing of current
    - cardio-respiratory arrest
        - causes include:

                - current induced dysrhythmia (VF or VT)
                - hypoxic cardiac arrest
                - hypovolaemia from associated injuries
                - hyperkalaemia from rhabdomyolysis
            - avoid IV cannulation of an involved limb
                - vascular structures may be damaged by current
    - thermal injury
        - examination of entrance and exit burns to appreciate structures potentially involved
        - administration of large volumes of fluid may be required
            - haemodilution may cause requirement for blood transfusion to maintain adequate circulating haemoglobin
        - traditional burns resuscitation formulae may lead to underestimation of fluid requirements
        - early invasive monitoring can be useful to help guide fluid requirements
        - analgesia
        - photography, dressing and where appropriate elevation of burns
        - plastics consultation
        - assess for compartment syndrome
            - can be difficult to distinguish from muscle ischaemia secondary to vascular injury
            - vascular injury can result in arterial thrombosis and distal ischaemia
                - regular assessment of distal pulses, limb temperature and pain
        - skeletal muscle damage can result in electrolyte derangement and myoglobin release
            - potassium, phosphate, calcium and CK should be checked and repeated if needed
            - false positive urine test for haemoglobin due to myoglobinuria
                - can cause renal tubular dysfunction
                - requires adequate circulating volume and blood pressure
                - may require haemofiltration
        - severely burned patients may develop SIRS and multi-organ dysfunction
- prognosis
    - patients with a domestic shock who have no visible injury and a normal ECG can be discharged
    - patients with arrhythmias should have prolonged monitoring
    - women more than 22 weeks pregnant should have obstetric assessment
    - electrical burns should be managed in the same way as other burns but with a lower threshold for admission and review as they tend to be deeper than pure thermal burns

TC4 Salter-Harris classification

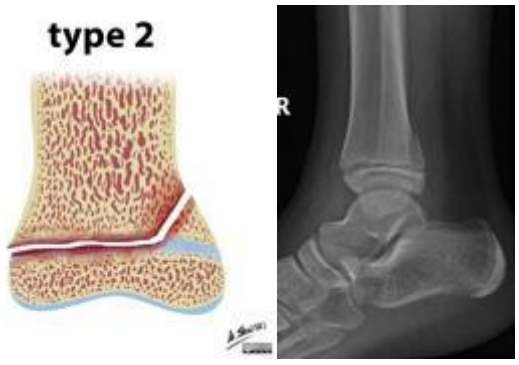
- background
    - describes injury to the epiphyseal plate of any bone
    - significant as they can cause growth issues
    - epiphysis

        - the end of a long bone
    - physis
        - the cartilaginous growth area of bone
    - metaphysis
        - the shaft of a long bone
- mnemonic
    - SALTR
        - **s**lipped/**s**eparated (I)
        - **a**bove (through metaphysis) (II)
        - **l**ower than (through epiphysis) (III)
        - **t**hrough/**t**ransverse (metaphysis and epiphysis) (IV)
        - **r**ammed (V)
- types
    - type I



https://radiopaedia.org/articles/salter-harris-classification?lang=gb

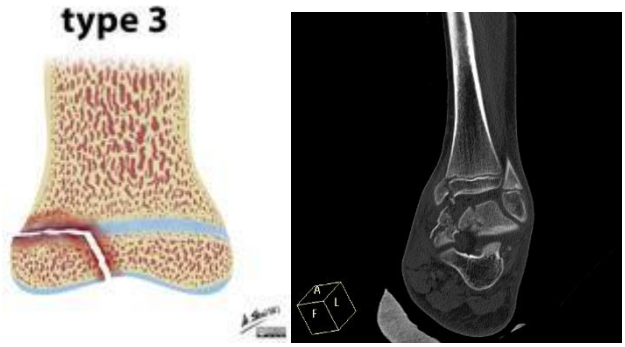
        - slipped/separated
        - 5-7%
        - fracture plane passes all the way through the growth plate, not involving bone
        - cannot occur if the growth plate is fused
        - there may be angulation, displacement and rotation
        - good prognosis
    - type II



https://radiopaedia.org/articles/salter-harris-classification?lang=gb

- above
- around 75% (most common)
- fracture passes across most of growth plate and up through metaphysis
    - a triangular fragment (the Thurston Holland fragment) is left intact
- up to half are at the distal radius, with other common sites distal tibia, distal fibula and phalanges
- good prognosis

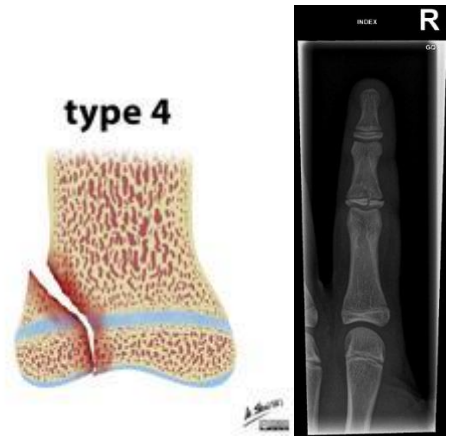
- type III



https://radiopaedia.org/articles/salter-harris-classification?lang=gb

- lower
- 7-10%
- fracture plane passes some distance along the growth plate and down through the epiphysis
- most often seen at the distal tibia and distal phalanx
- angulation, displacement and rotation may occur
- poorer prognosis as the proliferative and reserve zones are interrupted

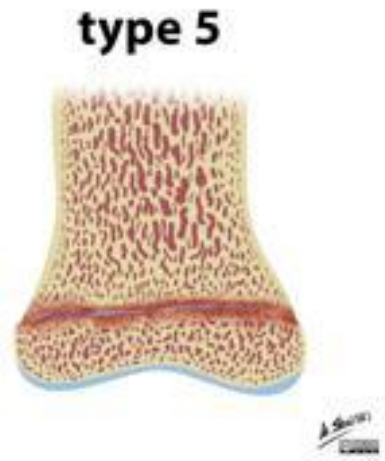
- type IV



https://radiopaedia.org/articles/salter-harris-classification?lang=gb

- through/transverse
    - intra-articular
    - 10%
    - fracture plane passes directly through the metaphysis, growth plate and down through the epiphysis
        - most common at distal radius, phalanges and distal tibia

- angulation, displacement and rotation may occur
    - poor prognosis as proliferative and reserve zones are interrupted
        - often lead to altered joint mechanics and functional impairment
        - orthopaedic evaluation and subsequent operative intervention often required
- type V



https://radiopaedia.org/articles/salter-harris-classification?lang=gb

    - rammed
    - uncommon (<1%)
        - usually identified retrospectively when clinical deformity or radiographic evidence of growth arrest are seen
    - crushing type of injury, does not displace growth plate but damages it by direct compression
    - worst prognosis
        - disruption of the germinal matrix and associated vascular supply lead to potential bone growth arrest, altered joint mechanics and functional impairment
        - orthopaedic evaluation and subsequent operative intervention often required
- uncommonly used Salter-Harris types
    - type VI
        - injury to the perichondral structures
    - type VII
        - isolated injury to the epiphyseal plate
    - type VIII
        - isolated injury to the metaphysis, with a potential injury related to endochondral ossification
    - type IX
        - injury to the periosteum that may interfere with membranous growth

TC5 infection – paronychia, pulp space, flexor sheath, nail bed, amputations etc.

- paronychia

https://dermnetnz.org/topics/paronychia/

- definition
  - inflammation of the skin around a finger or toenail
  - can be acute or chronic
- risk factors
  - nail biting/picking at the nail fold
  - sucking fingers or thumbs
  - manicures
  - ingrowing toenails
  - sculpted or artificial fingernail application
  - oral retinoid treatment – dries the skin
  - chronic paronychia is common in people with hand dermatitis or constantly cold, wet hands such as dairy farmers, fishermen, bartenders, cleaners
- cause
  - usually bacterial infection
    - Staph aureus
    - Strep pyogenes
    - Pseudomonas
  - can be viral
    - e.g. herpes simplex
  - can be fungal
    - e.g. Candida albicans
  - causes of chronic paronychia are not well understood
- clinical features
  - acute paronychia
    - develops rapidly over a few hours
    - usually affects single nail fold
    - pain, redness, swelling
    - if herpes simplex is the cause there may be multiple tender vesicles
    - pus can appear under the cuticle and evolve to an abscess
    - if caused by Strep pyogenes there can be fever, lymphangitis and tender lymphadenopathy
  - chronic paronychia
    - may start in one nail fold but often spreads to several other fingers
    - each nail fold is swollen and lifted off the nail plate

                - skin may be occasionally red and tender
                - there is sometimes pus under the cuticle
                - the nail plate thickens and distorts, often with transverse ridges
        - complications
            - cellulitis
            - infectious tendonitis
            - nail dystrophy with chronic paronychia
                - takes up to a year for the nails to return to normal after recovery
        - diagnosis
            - clinical
            - may have supporting laboratory evidence:
                - gram stain and microscopy
                - bacterial culture
                - viral swabs
                - nail clippings for culture
        - management
            - acute paronychia
                - soak affected digit in warm water several times a day
                - consider topical antiseptic for localised, minor infection
                - if infection severe or prolonged, oral antibiotics – e.g. doxycycline
                - consider acyclovir for severe herpes simplex infection
                - incision and drainage of any abscess with irrigation and gauze packing
                - occasional the nail needs to be removed to drain pus
            - chronic paronychia
                - keep hands dry and warm
                - avoid wet work or use waterproof gloves lined with cotton
                - keep fingernails clean
                - wash with soap and water after dirty work, rinse and dry carefully
                - frequent emollient hand cream
                - treatment of any microbes grown on culture
                - topical corticosteroid ointment for 2-4 weeks
                - consider tacrolimus ointment, intralesional steroid injections, antiseptics, antifungals
- pulp space infection

https://www.orthobullets.com/hand/6102/felon

    - also called a felon

- incidence
    - 15-20% of hand infections
- anatomical location
    - usually thumb or index finger
- anatomy
    - fingertip pulp is a closed sac with connective tissue framework separated by fibrous vertical septae running from the periosteum of the distal phalanx to the epidermis
        - provides structural support
        - stabilises the pulp during pinch and grasp
    - pulp contains eccrine sweat glands that open onto the epidermis
    - blood supply
        - digital arteries run parallel to the distal phalanx
            - give off a nutrient branch to the epiphysis before entering the pulp space
- mechanism
    - penetrating injury
        - blood glucose needlestick
        - splinters
    - local spread
        - from paronychia
    - no history of injury in 50%
        - may result from bacterial contamination of the fat pad through the eccrine sweat glands
- pathophysiology
    - swelling and pressure within micro-compartments leads to ‘compartment syndromes’ of the pulp
    - inflammation and cellulitis leads to local vascular congestion
    - if untreated, tissue necrosis and abscess formation leading to increased microvascular impairment
    - increasing pressure can lead to ischaemia
        - affects blood supply to diaphysis more than to the skin causing bone necrosis and sequestration before spontaneous decompression through the skin
    - organisms
        - Staph aureus
            - most common
        - Gram negative organisms
            - in immunosuppressed patients
        - Eikenella corrodens
            - in diabetics who bite their nails
- symptoms
    - severe throbbing pain
- signs
    - swelling
        - does not extend proximal to DIP flexion crease unless flexor tendon sheath or joint is involved

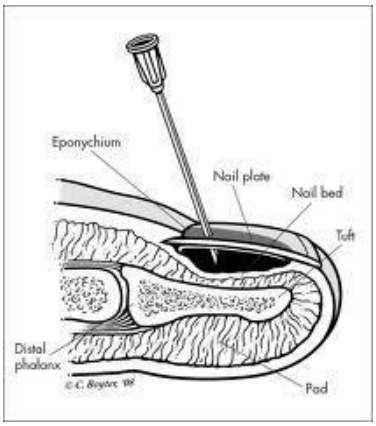
        - tenderness
    - investigations
        - x-ray only if history of trauma or foreign body
    - management
        - non-operative
            - oral antibiotics and observation
            - for early felon with no drainable abscess
        - operative
            - bedside incision and drainage with IV antibiotics
                - required in most cases
                - pus should be sent for culture, wound packed and dressed with irrigation and regular repacking
                - involves digital nerve block and incision distal to DIP crease to prevent contracture and extension into the flexor sheath
                - volar longitudinal approach for most direct access
                    - for superficial felons, foreign body penetration or visible drainage mid-lateral approach with incision on ulnar side for digits 2-4 and radial side for thumb and digit 5
                    - for deep felons with no foreign body and no drainage
    - complications
        - finger tip compartment syndrome
        - flexor tenosynovitis
        - osteomyelitis
        - digital tip necrosis
- flexor sheath infection (pyogenic flexor tenosynovitis)

https://www.orthobullets.com/hand/6105/pyogenic-flexor-tenosynovitis

    - incidence
        - 2.5-9.4% of all hand infections
    - risk factors
        - diabetes
        - IV drug use
        - immunocompromise

- pathophysiology
  - mechanism
    - penetrating trauma to the tendon sheath
    - direct spread from:
      - felon
      - septic joint
      - deep space infection
    - infection travels in the synovial sheath that surrounds the flexor tendon
  - microbiology
    - Staph aureus
      - most common (40-75%)
    - MRSA
      - 29%
      - intravenous drug users
    - other common skin flora
    - mixed flora and Gram negative organisms
      - immunocompromised patients
    - Eikenella
      - human bites
    - Pasteurella multocida
      - animal bites
- anatomy
  - tendon sheaths
    - protect and nourish the tendons
    - variations in anatomy are common
- symptoms
  - pain and swelling
    - usually delayed fashion over 24-48 hours
    - usually localised to palmar aspect of one digit
- examination
  - Kanavel signs
    - flexed posturing of the involved digit
    - tenderness to palpation over the tendon sheath
    - marked pain with passive extension of the digit
    - fusiform swelling of the digit
  - increased warmth and erythema of the involved digit
- imaging
  - not usually required unless to rule out foreign body
  - MRI may determine the extent
- management
  - non-operative (rare)
    - only for early presentation
    - hospital admission, IV antibiotics, hand immobilisation, observation
    - surgery not required if signs of improvement within 24 hours
  - operative
    - incision and drainage followed by culture-specific IV antibiotics

- low threshold for operative management
    - complications
        - stiffness
        - tendon or pulley rupture
        - spread of infection
        - loss of soft tissue
        - osteomyelitis
- nail bed injury
    - background
        - sharp injuries that penetrate the nail bed tend to continue through the bony phalanx and cause fingertip amputation; the majority of fingertip injuries are crush injuries
            - crush injuries cause the nail plate to buckle and compress the nail plate against the terminal phalanx
    - subungual haematoma
        - small nail bed laceration occurs and blood cannot drain from an intact nail
        - causes severe pain
        - small haematomas can be drained by decompression with a hot paper clip or battery-powered cautery



https://www.orthobullets.com/hand/6109/nail-bed-injury

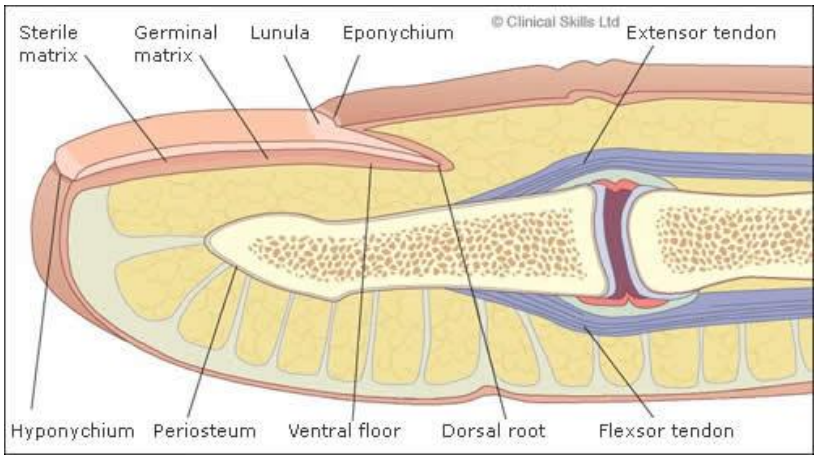
        - large haematomas where the nail plate is not adherent to the nail bed may require nail removal and nail bed repair
    - nail bed laceration
        - should be suspected in all fingertip crush injuries
        - simple or stellate nail bed lacerations without underlying fracture have a better prognosis than nail bed avulsions
        - managed with nail removal and nail bed repair
            - the nail plate is separated and removed from the nail bed with fine scissors
            - the nail bed is repaired with fine absorbable sutures such as Vicryl Rapide 6.0
                - Dermabond may be considered as an alternative
            - repair should include the dorsal roof and ventral floor of the nail fold

- the removed nail is trimmed of sharp edges and replaced in the nail fold to act as a stent for the nail bed, a template for the new nail and a protective cover to reduce pain and discomfort
- a transverse suture through the nail and lateral folds helps retain it in position
- nail is retained for around 4 weeks before discarding by cutting the retaining sutures

▪ nail bed laceration with associated fracture
- nail bed is supported internally by the terminal phalanx bone and externally by the nail plate
- if there is a stable undisplaced fracture of the terminal phalanx bone not requiring internal fixation, simple nail bed repair with external splintage is required
- displaced or unstable fractures require referral for specialist treatment as they require accurate reduction and internal stabilisation, usually K-wiring, before nail bed repair
  - residual dorsal step deformity of the terminal phalanx bone can result in nail bed irregularity, scarring, nail plate detachment and late nail deformity

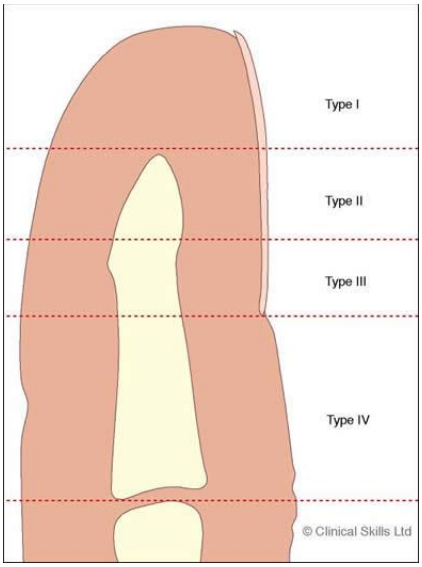
o nail bed avulsion
▪ usually avulsion of all or part of nail and portion of underlying nail bed
▪ usually caused by higher energy injuries
▪ can be associated with distal phalanx fractures
▪ managed with nail bed removal, nail bed repair +/- fracture fixation
▪ if significant loss of nail matrix, may require nail matrix transfer from adjacent finger
▪ tetanus booster if required, IV antibiotics
▪ these injuries should be referred to a hand specialist

- amputation
  - background
    - the fingertip is the part of the distal phalanx distal to the insertion of extensor and flexor tendons
    - fingertip anatomy



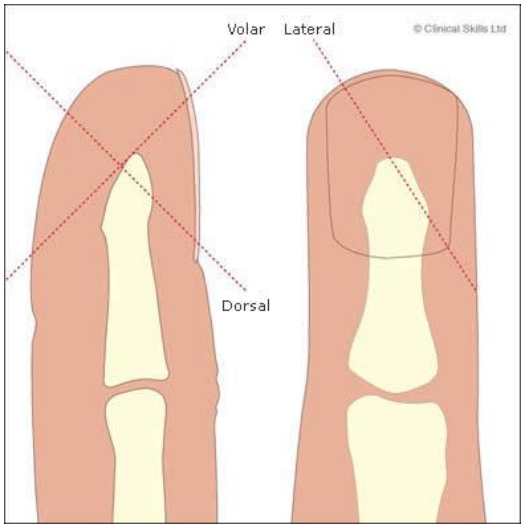
https://www.rcemlearning.co.uk/reference/soft-tissue-injuries-of-the-hand/#1583314730191-17dddd2c-5a2e

        - eponychium
            - soft tissue on the dorsal surface just proximal to the nail
        - paronychium
            - lateral nail folds
        - hyponychium
            - plug of keratinous material situated beneath the distal edge of the nail where the nail bed meets the skin
        - lunula
            - white portion of the proximal nail
            - demarcates the sterile from the germinal matrix beneath
        - nail bed
            - sterile matrix
                - where the nail adheres to the nail bed
            - germinal matrix
                - proximal to the sterile matrix
                - responsible for 90% of nail growth
    - transverse amputations
        - partial amputations often seen in crush injuries
        - classification:
            - type I
                - fingertip soft tissue loss only
            - type II
                - fingertip loss at the level of the proximal third of the nail plate
            - type III
                - fingertip loss at the level of the eponychial fold
            - type IV
                - fingertip loss proximal to the DIP joint



https://www.rcemlearning.co.uk/reference/soft-tissue-injuries-of-the-hand/#1583314730191-17dddd2c-5a2e

    - oblique fingertip defects
        - classified as volar, dorsal or lateral



https://www.rcemlearning.co.uk/reference/soft-tissue-injuries-of-the-hand/#1583314730191-17dddd2c-5a2e

- injuries suitable for ED management
  - superficial skin loss with defect <1cm^2 in zone 1
  - transverse type I fingertip amputations
  - type I and type II fingertip amputations in children
  - oblique partial fingertip amputation without bone exposure
  - stable fracture needing splintage only
- follow-up clinic, dressings facilities and access to physiotherapy are required
- procedure for ED management
  - local anaesthesia ring block
  - little finger of size glove with tip cut off can be used as finger tourniquet to exsanguinate the digit
  - thorough cleaning, removing dirt and foreign material
  - trim off devitalised tissue
  - non-adherent dressings
    - dressings changed and wound inspected 2-3 times/week
  - a stable fracture of the terminal phalanx may be externally splinted for 2-3 weeks
  - a 1cm^2 defect takes an average of 5 weeks to heal
- injuries for referral to hand specialist
  - type II or higher partial amputation of fingertip
  - oblique fingertip partial amputation with bone exposure or fracture
  - large soft tissue defect >1cm^2
  - unstable fracture of terminal phalanx
- the amputated part should not be discarded until the wound has been fully examined
  - it may be possible to use skin from it as a graft to cover the open wound
- specialist surgical options
  - bone shortening and primary skin closure
  - tip reposition
  - local skin flap
  - regional skin flap
  - distant skin flap

        - skin graft
        - toe pulp transfer
        - replantation
        - microvascular flap
    - patient preparation prior to transfer to hand specialist
        - IV fluids
            - if patient fasting for general anaesthetic
        - IV antibiotics
            - e.g. first generation cephalosporin
        - analgesia
            - IV opiate or digital block
        - x-ray
            - of the digit and the amputated segment if available
        - clean and dress finger stump
            - non-adherent dressing
            - stump wrapped lightly with sterile dressings and bandage
        - elevate hand in sling
        - tetanus prophylaxis
        - care of amputated part
            - remove any foreign material from exposed soft tissues
            - clean amputated part with saline
            - wrap in damp (not wet) saline moistened gauze
            - place wrapped segment in plastic bag
            - place bag in a container filled with ice mixed with saline
                - amputated part should not be directly on ice
            - ensure bag is appropriately labelled with patient details and part

TC6 animal bites including human

- dog and cat bites
    - epidemiology
        - dog bites are most common animal bites, then cats, then humans
        - more common in males
        - more common from ages 2-19 years
        - more common on the upper extremities
    - pathophysiology
        - dog bites
            - cause crush wounds, puncture wounds, avulsions, tears and abrasions
            - large dogs exert significant pressure from their jaws
            - more likely to cause structural damage to vessels, nerves and joints
            - microbiology
                - Pasteurella (50% of dog bites)
                    - Pasteurella carnis
                - Staph aureus
                - alpha-haemolytic Strep
                - Corynebacterium
                - anaerobes (e.g. Bacteriodes)

        - Capnocytophaga canimorsus
            - rare, potentially fatal (in splenectomy patients)
            - causes cellulitis, sepsis, endocarditis, meningitis, DIC, ARDS, death
            - highest mortality in immunocompromised patients (30-60%)
    - cat bites
        - penetrate bones and joints
            - cause septic arthritis and osteomyelitis
        - small sharp teeth cause puncture wounds that seal immediately
        - penetrate joints and flexor tendons
        - higher risk of infection than dog bites
        - microbiology
            - Pasteurella (70-80%)
                - Pasteurella multocida and septica
                - causes intense pain and swelling in 48 hours
            - other organisms similar to dog bites
    - risk factors for secondary bacterial infection
        - bite to hand, foot or major joint
        - puncture wounds or crush injuries
        - treatment delay >12 hours
        - age >50 years
        - pre-existing host disease
            - immunosuppression (e.g. steroids, asplenism)
            - chronic alcoholism
            - diabetes mellitus
            - vascular disease
            - existing oedema of extremity
    - rabies (if bitten in area where it is transmitted)
        - caused by a rhabdovirus
        - common animal carriers include dogs, raccoons, bats, foxes
        - increased risk with open wounds, scratches, abrasions, mucous membrane involvement
- history
    - type of animal
    - time of injury
    - co-morbidities
- symptoms
    - pain and swelling
    - bleeding
    - symptoms of local or systemic infection
- examination
    - evaluate depth of wound and presence of crush injury
    - check neurovascular status
    - look for joint penetration
    - photography of wounds
- x-ray indications

        - crush injuries
        - suspected fracture
        - suspected foreign body
    - management
        - non-operative
            - copious irrigation
                - >150ml saline irrigation through needle or plastic catheter
            - indications for antibiotics (co-amoxiclav, cefuroxime, ceftriaxone)
                - cat bites
                - presentation >8 hours
                - immunocompromised or diabetic
                - hand bite
                - deep bites
            - immobilisation and elevation
        - operative
            - surgical debridement
                - indications:
                    - crush or devitalised tissue
                    - foreign body
                    - bites to digital pulp space, nail bed, flexor tendon sheath, deep spaces of the palm, joint spaces
                    - tenosynovitis
                    - septic arthritis
                    - abscess formation
    - prognosis
        - serious and fatal bites include:
            - large aggressive dogs
            - small children
            - head and neck bites
- human bites
    - background
        - associated with infections of the hand due to presence of anaerobic and aerobic pathogens
        - diagnosis made by history and presence of open wounds, most commonly over the dorsal aspect of the 3rd or 4th MCP joint
        - treatment is generally surgical debridement and antibiotics
    - epidemiology
        - human bite wounds to the hand are approximately 2% of bite wounds
        - more common in males
    - pathophysiology
        - mechanism
            - usually direct clenched fist trauma from tooth after punching someone in the mouth
            - can result from direct bite
        - pathoanatomy
            - the tooth penetrates the capsule of the MCP joint
            - flora from the mouth enter the joint

- bacteria become trapped within the joint as the fist is released from a clenched position
    - bacteria are caught under the extensor tendon and/or capsule
- microbiology
    - typically polymicrobial
    - most common organisms:
        - alpha-haemolytic Streptococcus (S. viridans) and Staphylococcus aureus
        - Eikonella corodens (7-29%)
        - other Gram negative organisms
- associated conditions
    - extensor tendon lacerations
        - can be missed due to proximal tendon retraction

- history
    - direct clenched-fist trauma to another individual's mouth
        - often overlooked
        - high index of suspicion required as patients may be reluctant to reveal history
        - consider the injury a 'fight bite' until proven otherwise
    - may be delay in presentation until symptoms become intolerable
- symptoms
    - progressive development of pain, swelling, erythema and drainage over wound
- examination
    - small wound over dorsal aspect of MCP joint
        - often transverse and irregular
        - typically 3rd and/or 4th MCPs, can involve any digit
        - erythema, warmth and/or oedema overlying the wound and joint
        - may be purulent drainage
    - assess for integrity of extensor tendon function
    - possible pain with passive ROM of MCP joint
    - typically no involvement of volar/flexor surface of digit
    - neurovascular status typically preserved
- x-rays
    - to assess for foreign body (i.e. tooth fragment) or fracture
- management
    - for those not requiring referral: antibiotic prophylaxis for bites that have broken the skin and not drawn blood
        - co-amoxiclav, co-trimoxazole or doxycycline
        - also for wounds that have broken the skin but not drawn blood if:
            - high-risk area such as hands, feet, face, genitals, skin overlying cartilaginous structures, areas of poor circulation
            - person at risk of serious infection due to co-morbidities
    - incision and drainage, IV antibiotics
    - indications:
        - fight bite

                - joints or tendon sheaths involved
            - antibiotics
                - IV antibiotics to cover Staph, Strep and Gram negative organisms
                - PO antibiotics (co-amoxiclav) on discharge for 7 days)
            - debridement
                - debridement of wound and joint capsule
                - wound left open for drainage
                - Gram stain and culture
- reasons for referral to secondary care (NICE recommendations)
    - severe bite injuries with heavy bleeding causing haemodynamic instability
    - penetrating wounds involving, arteries, joints, nerves, muscles, tendons, bones or the central nervous system
    - facial wounds (excluding very minor wounds) and bites to the eye or orbit
    - bites with a possibility of a foreign body (e.g. tooth) in the wound
    - devitalised wounds requiring debridement
    - wounds that may benefit from closure
    - injuries requiring reconstructive surgery
    - people with abscesses, severe cellulitis, lymphangitis, osteomyelitis, septic arthritis, necrotising fasciitis, infected bite wounds not responding to treatment or systemically unwell with possible sepsis
    - people with increased risk of infection (e.g. immunosuppression)
    - bites to poorly vascularised areas such as ear or nose cartilage
    - people who cannot take oral antibiotics (preferably having explored community parenteral options first)
    - people who have developed signs or symptoms of severe infection whilst on antibiotics
    - safeguarding concerns
    - requirement for tetanus immunoglobulin
    - possible exposure to BBV (should be discussed with infectious diseases team)
- assessment of tetanus risk
    - immunisation status
    - determine if the injury is likely to be a tetanus-prone wound, e.g.:
        - delay in surgical intervention for more than 6 hours
        - significant amount of devitalised tissue or puncture wound (especially if in contact with soil or manure)
        - a foreign body in the wound
        - compound fracture
        - systemic sepsis
    - from government Green Book:
        - tetanus
            - notifiable disease
            - caused by spores from Clostridium tetani
            - present in soil or manure, can be introduced into the body through a puncture wound, burn or scratch
            - incubation period 4-21 days
            - primary immunisation

    - 3 doses of tetanus-containing vaccine (usually DTaP), usually at two, three and four months
        - can be given at any time between 2 months and 10 years
    - for children over 10 years/adults, 3 doses of suitable vaccine (usually Td/IPV) with an interval of one month between each dose
- boosters
    - first booster should be three years after the primary course
    - second booster 10 years after the first booster
- after a clean wound, no booster required but if person not fully immunised opportunity can be taken to start to complete course
- tetanus-prone wounds:
    - puncture-type injuries acquired in a contaminated environment likely to contain tetanus spores (e.g. gardening injuries)
    - wounds containing foreign bodies
    - compound fractures
    - wounds or burns with systemic sepsis
    - certain animal bites or scratches
        - animal saliva from domestic pets should not contain tetanus spores unless the animal has been routing in soil or lives in an agricultural setting
- high risk tetanus-prone wounds are any of the above with:
    - heavy contamination with material likely to contain tetanus spores (e.g. soil, manure)
    - wounds or burns with extensive devitalised tissue
    - wounds or burns that require surgical intervention that is delayed for more than six hours (even if contamination not initially heavy)
- immediate treatment
    - for children under 5 who have completed initial course, children 5-10 who have had initial course and first booster and those over 11 who have had a priming course with last dose within 10 years
        - no immediate treatment required for any type of wound
    - for children 5-10 who have received priming course but no booster and adults who have received adequate priming course with last booster >10 years ago (anyone born in the UK after 1961 with no history of refusing vaccines)
        - clean wound: none required
        - tetanus-prone wound: immediate reinforcing dose of vaccine
        - high risk tetanus-prone wound: immediate reinforcing dose of vaccine and one dose of human tetanus immunoglobulin at a different site

        - for patients without adequate priming course (including uncertainty and/or born before 1961)
            - clean wound: immediate reinforcing dose of vaccine
            - tetanus-prone wound: immediate reinforcing dose of vaccine and one dose of human tetanus immunoglobulin at a different site
            - high risk tetanus-prone wound: immediate reinforcing dose of vaccine and one dose of human tetanus immunoglobulin at a different site
            - further later doses as required via GP
    - doses:
        - 250 IU tetanus immunoglobulin IM
        - 500 IU if >24 hours since injury, risk of heavy contamination or following burns
        - vaccines are in pre-dosed syringes

TC7 injury to bladder, urethra, testes or penis

- background
    - most genitourinary injuries can undergo delayed repair once patient stabilised and other injuries dealt with
    - renal pedicle injury is an exception
        - can lead to life-threatening haemorrhage and renal ischaemia
        - typically results from deceleration, leading to the kidney swinging violently on its vascular pedicle
        - often requires nephrectomy
- haematuria
    - severe injuries such as renal artery injury or uretopelvic disruption can present without haematuria
    - 5% of renal injuries and 20% of renovascular injuries lack haematuria
    - in general the greater the degree of haematuria the greater the risk of significant intra-abdominal injury
    - management of microscopic haematuria following blunt abdominal trauma
        - asymptomatic:
            - no imaging required (extremely low yield)
            - arrange repeat urinalysis (e.g. in a week's time) and close follow up by GP
            - some experts advise imaging in asymptomatic children with blunt trauma and microscopic haematuria as they are more vulnerable to significant renal injury
        - symptomatic
            - CT abdomen with contrast
    - management of macroscopic haematuria following blunt abdominal trauma
        - CT abdomen with IV contrast and CT cystogram
            - 50% of patients have renal injuries, a further 15% have injuries to other intra-abdominal injuries
        - retrograde urethrogram also required if urethral injury suspected
        - character of urine is suggestive (but not diagnostic) of injury type

- faint haematuria
    - shades of pink
    - renal injury or bladder contusion
- moderate amount of darkly bloody urine
    - frequently associated with extraperitoneal bladder injury
- small amount of very dark, bloody urine
    - frequently associated with extraperitoneal bladder injury
- scant and very dark blood in the catheter
    - suggests urethral injury or catheter balloon inflated in the urethra

- increasing urea and creatinine in patients with urinary tract trauma
    - most likely caused by:
        - renal impairment due to traumatic injury
        - reabsorption of extravasated urine
        - contrast induced nephropathy following diagnostic imaging
- skeletal injuries associated with genitourinary tract trauma
    - pelvic fractures
        - posterior urethral injury (above the urogenital diaphragm)
        - bladder injury
    - perineal straddle injury
        - anterior urethral injury
    - fracture of the lower posterior ribs, lower thoracic or lumbar vertebrae
        - renal or ureteral injuries
- kidney injury
    - aetiology
        - blunt trauma from motor vehicle collisions, falls etc most common cause
            - deceleration injuries with collision of the kidney with the vertebral column or ribcage
        - 95-98% are minor injuries
    - types of kidney injury
        - contusion/haematoma
        - laceration
        - haemorrhage
        - avulsion of renal pedicle leading to devascularisation of the kidney
        - pseudoaneurysm
        - AV fistula
        - renal artery thrombosis, transection or dissection
    - recognition
        - clinically significant injuries will have at least one of:
            - macroscopic haematuria
            - loin tenderness and/or swelling
            - haemodynamic instability
    - fracture of the lower posterior ribs, lower thoracic or lumbar vertebrae may be present
    - investigation
        - CT abdomen with IV contrast
        - IV pyelogram

- if CT unavailable or imaging needs to be carried out in the operating theatre
- less sensitive
- does not visualise non-urological injuries

- renal angiography
  - rarely required

- AAST renal trauma grading
  - grade I
    - subcapsular haematoma or contusion, without laceration
  - grade II
    - superficial laceration ≤1cm depth not involving the collecting system (no evidence of urine extravasation)
    - perirenal haematoma confined within the perirenal fascia
  - grade III
    - laceration >1cm not involving the collecting system (no evidence of urine extravasation)
    - vascular injury or active bleeding confined within the perirenal fascia
  - grade IV
    - laceration involving the collecting system with urinary extravasation
    - laceration of the renal pelvis and/or complete ureteropelvic disruption
    - vascular injury to segmental renal artery or vein
    - segmental infarctions without associated active bleeding (i.e. due to vessel thrombosis)
    - active bleeding extending beyond the perirenal fascia (i.e. into retroperitoneum or peritoneum)
  - grade V
    - shattered kidney
    - avulsion of renal hilum or laceration of the main renal artery or vein; devascularisation of the kidney due to hilar injury
    - devascularised kidney with active bleeding
- treatment and prognosis by grade
  - grade I
    - conservative management
  - grade II
    - conservative management under close observation
  - grade III
    - conservative management under close observation
    - may be managed surgically if undergoing laparotomy for other abdominal injuries
  - grade IV
    - surgical management, especially if undergoing laparotomy for other abdominal injuries
  - grade V
    - surgical management
    - often require nephrectomy

        - interventional radiology and stenting may sometimes be considered
    - complications (affect around 7.5%)
        - urinoma (most common)
            - encapsulated urine collection due to urinary leakage
            - treated conservatively if small
            - if large, recurrent or symptomatic may be managed with percutaneous drainage
        - delayed bleeding (within 1-2 weeks of injury)
        - urinary fistula
        - perinephric abscess
        - hypertension from renal artery injury
        - hydronephrosis
        - pyelonephritis
- urethral injury
    - recognition
        - more common in males due to urethra being 4-5 times longer than in females
        - associated with displaced fractures of the pelvic ring
        - gross haematuria, difficulty placing a urinary catheter
        - classic clinical features are uncommon:
            - blood at the meatus
            - perineal/scrotal haematoma
            - high riding prostate on examination
        - inability to void in complete urethral disruption
    - causes
        - blunt trauma (shearing/straddle injuries)
            - posterior urethral injury is caused by crushing force to the pelvis
                - associated with pelvic fractures and bladder injury
            - anterior urethral injury is usually caused by a straddle injury and is an isolated injury
        - penetrating trauma (e.g. stab wounds, gunshot wounds, dog bites)
            - more commonly affect the anterior urethra
        - iatrogenic
            - catheterisation, removal of catheter without deflating balloon, cystoscopy
            - post-surgical
    - retrograde urethrogram should be performed prior to urinary catheterisation but after major pelvic bleeding has been excluded (contrast may interfere with scan interpretation)
        - however it is probably safe to perform careful, gentle catheterisation
    - classification (Goldman classification)
        - type I
            - stretching of the posterior urethra due to disruption of puboprostatic ligaments, though the urethra is intact
        - type II
            - posterior urethral injury above urogenital diaphragm
        - type III

                - injury to the membranous urethra, extending into the proximal bulbous urethra (i.e. with laceration of the urogenital diaphragm)
            - type IV
                - bladder base injury involving bladder neck extending into the proximal urethra
                - potential for incontinence if internal sphincter is injured
            - type Iva
                - bladder base injury but not involving bladder neck (cannot be differentiated from type IV radiographically)
            - type V
                - anterior urethral injury (isolated)
        - treatment and prognosis
            - treatment ranges from urinary diversion (suprapubic catheter) to primary or delayed urethral anastomosis depending on severity
            - most common long term complication is urethral stricture
- bladder injury
    - recognition
        - usually associated with pelvic fractures
            - macroscopic haematuria in a patient with pelvic trauma requires investigation
        - penetrating trauma close to the bladder
        - classic triad:
            - haematuria
            - suprapubic pain
            - inability to void
        - pregnant women and intoxicated patients are at higher risk
    - investigation
        - retrograde cystogram or CT cystography
            - contrast injected via urethral catheter
            - should be delayed until life-threatening pelvic injuries are excluded or stabilised
    - categorisation
        - contusion or haematoma
        - intraperitoneal rupture
        - extraperitoneal rupture
    - management
        - bladder contusion and haematoma can be observed
        - intraperitoneal rupture requires laparotomy and surgical repair
        - extraperitoneal rupture can often be managed with simple catheterisation (about 10 days)
- penile fracture
    - rupture of the corpus cavernosus due to a tear in the tunica albuginea
    - usually caused by vigorous uncoordinated sexual intercourse but can be caused from a fall or direct trauma
    - suggestive history is sudden detumescence following a loud crack at the time of the trauma
    - there is penile haematoma and difficulty in voiding

    - urethral and corpus spongiosum injury (5-20%) suggested by:
        - blood at the meatus
        - inability to pass urine
        - extravasation of urine
    - management is surgical repair
- penile amputation
    - apply pressure to bleeding stump, provide analgesia, wrap the amputated part in dry sterile gauze before placing in ice (avoid direct contact of the amputated part with ice)
    - management is surgical reimplantation (ideally <6 hours warm ischaemic time) or reconstruction
- scrotal injury
    - main concern is testicular rupture
    - recognition
        - blunt trauma to the scrotum such as a kick or fall
        - scrotal haematoma and tenderness
    - investigation
        - scrotal ultrasound
    - management
        - a dislocated (luxed) testicle can be reduced in the ED (replaced into the scrotal sac)
        - surgical repair for testicular rupture, haematocoele, non-reducible testicular dislocation and scrotal degloving

UROLOGY

UP1 dysuria

- causes
    - abdominal (due to irritation of nearby urinary structures)
        - appendicitis
        - ectopic pregnancy
    - urinary tract
        - UTI
            - bacterial UTI
            - urethritis
                - e.g. chlamydia, gonococcus or non-gonococcal
                - common cause of dysuria in men <35
            - urinary schistosomiasis
        - interstitial cystitis
        - obstruction
            - prostatic enlargement
            - urethral stricture
        - kidney stones in bladder or urethra
        - malignancy
            - e.g. bladder carcinoma, urethral tumour
    - genital causes

        - urethral or vaginal trauma
            - including sexual abuse or foreign body
        - genital herpes simplex
        - women
            - vaginitis
                - vaginal candidiasis
                - atrophic vaginitis
                - bacterial vaginosis
        - men
            - prostatitis
            - epididymo-orchitis
            - epididymitis
    - other disease
        - spondyloarthropathy
            - reactive arthritis
            - Behçet's disease
        - compression from pelvic mass
    - irritants
        - drugs
            - cyclophosphamide
            - allopurinol
            - danazol
            - tiaprofenic acid
            - possibly other NSAIDs
        - chemical irritants
            - allergic or irritant reaction to soaps, vaginal lubricants, spermicides, contraceptive foams and sponges, tampons, toilet paper
        - mechanical irritation
            - e.g. from poorly fitting contraceptive diaphragm or vaginal ring pessary
        - radiation or chemical exposure
- assessment
    - history
        - pain symptoms
            - onset and duration of dysuria
            - whether there is abdominal pain
                - consider appendicitis or ectopic pregnancy
            - radiation
                - e.g. to loin or back
        - other symptoms
            - fever, rigors or malaise
                - suggest pyelonephritis
            - haematuria
                - infection, stones, neoplasm, renal disease
            - urethral or vaginal discharge
                - genital tract infection
            - odour

        - possible bacterial infection
    - pruritus
        - common with genital candidiasis
    - frequency and urgency
        - bladder irritation
    - urine volume and flow
        - consider obstruction
- medical history
    - possible pregnancy
    - past history
        - previous UTI
        - other genitourinary disease
        - pelvic surgery or irradiation
        - other general illness
        - medication
- recent sexual history
    - method of contraception
    - consider possibility of childhood sexual abuse
- occupation
    - exposure to dyes and solvents as risk for bladder cancer

- examination
    - fever, tachycardia and loin tenderness
        - pyelonephritis
    - abdominal/pelvic tenderness; guarding; masses; adnexal tenderness; enlarged bladder
    - vaginal discharge, candidiasis, genital herpes simplex, vaginitis
    - enlarged prostate on rectal examination

- investigations
    - generally required for children and men, not necessarily for women
    - includes:
        - urine dipstick, microscopy and culture
        - pregnancy test if needed
        - referral to STI clinic for investigation
        - ultrasound of the urinary tract, pelvis or abdomen if there is suspicion of obstruction or masses
        - x-ray KUB or CT KUB if renal tract stones suspected
        - urodynamic studies
        - urine cytology
        - further specialist tests such as cystoscopy

UP2 injury to bladder, urethra, testes or penis

- background
    - most genitourinary injuries can undergo delayed repair, with the exception of renal pedicle injury
        - renal pedicle injury can lead to life-threatening haemorrhage and renal ischaemia

                - typically results from deceleration when the kidney swings violently on the vascular pedicle
                - can lead to thrombosis or complete detachment at the pedicle
                - early surgical repair is needed to rescue the kidney but nephrectomy is often required
        - significant injury including renal artery injury and ureteropelvic disruption can present without haematuria
            - around 5% of renal injuries and 20% of renovascular injuries lack haematuria
            - in general the greater the degree of haematuria, the greater the risk of significant intra-abdominal injury
- causes of rising urea and creatinine in patients with isolated urinary tract trauma
    - renal impairment due to traumatic injury
    - reabsorption of extravasated urine
    - contrast induced nephropathy following diagnostic imaging
- skeletal injuries associated with genitourinary trauma
    - pelvic fractures
        - posterior urethral injury (above the urogenital diaphragm)
        - bladder injury
    - perineal straddle injury
        - anterior urethral injury
    - fracture of the lower posterior ribs, lower thoracic or lumbar vertebrae
        - renal or ureteral injuries
- microscopic haematuria following blunt abdominal trauma
    - if the patient is asymptomatic, the yield of injuries requiring intervention is very low
        - no further imaging is needed
        - arrange repeat urinalysis (e.g. in a week) and GP follow up
    - some experts advocate imaging in children with asymptomatic haematuria following trauma as they are more vulnerable to significant renal injury
    - if the patient is significantly symptomatic, they may have associated non-urinary intra-abdominal or retroperitoneal injury
        - symptomatic patients should have a CT abdomen with contrast
- macroscopic haematuria following blunt abdominal trauma
    - CT abdomen with IV contrast and CT cystogram
        - 50% have renal injuries and a further 15% have injuries to other intra-abdominal organs
        - retrograde urethrogram should also be performed if urethral injury is suspected
    - character of bloody urine is suggestive but not diagnostic:
        - faint haematuria, primarily shades of pink
            - renal injury or bladder contusion
        - moderate amount of darkly bloody urine
            - extraperitoneal bladder injury
        - small amount of very dark, bloody urine
            - intraperitoneal bladder injury
        - scant and very dark blood in the catheter
            - urethral injury or catheter balloon inserted inside the urethra
- kidney injury

- recognition
  - clinically significant injuries will have at least one of:
    - macroscopic haematuria
    - loin tenderness and/or swelling
    - haemodynamic instability
  - fracture of the posterior ribs, lower thoracic or lumbar vertebrae may be present
  - CT abdomen with contrast is the investigation of choice
    - renal angiography is rarely required
  - injury grading
    - grade I
      - no laceration
      - subcapsular haematoma and/or contusion
    - grade II
      - superficial laceration ≤1cm depth not involving the collecting system (no evidence of urine extravasation)
      - perirenal haematoma confined within the perirenal fascia
    - grade III
      - laceration >1cm not involving the collecting system (no evidence of urine extravasation)
      - vascular injury or active bleeding confined within the perirenal fascia
    - grade IV
      - laceration involving the collecting system with urinary extravasation
      - laceration of the renal pelvis and/or complete ureteropelvic disruption
      - vascular injury to segmental renal artery or vein
      - segmental infarctions without associated active bleeding (i.e. due to vessel thrombosis)
      - active bleeding extending beyond the perirenal fascia (i.e. into the retroperitoneum or peritoneum)
    - grade V
      - shattered kidney
      - avulsion of renal hilum or laceration of the main renal artery or vein; devascularisation of a kidney due to hilar injury
      - devascularised kidney with active bleeding
- management
  - urology review
  - most renal injuries (grades I to III and most grade IV) can be managed conservatively and tend to heal spontaneously
  - surgical repair required for urinary extravasation or if ongoing bleeding or haemodynamic instability due to kidney injury
    - interventional radiology may be used to embolise bleeding vessels or stent dissected renal arteries
    - urinary extravasation may be amenable to stenting

        - grade V injuries (avulsed kidneys) need operative intervention and often require nephrectomy
- urethral injury
    - graded on a I-V scale and include contusion, stretch and partial or complete disruption
    - recognition
        - 95% males due to the urethra being 4-5 times longer than in females
        - associated with displaced fractures of the pelvic ring, particularly 'butterfly fractures'
        - gross haematuria, difficulty placing a urinary catheter
        - classic clinical features are uncommon:
            - blood at the meatus
            - perineal/scrotal haematoma
            - high riding prostate on examination
        - retrograde urethrogram should be performed prior to attempted catheterisation, but should be delayed until after the possibility of major pelvic bleeding has been excluded as any contrast extravasation will impact on the clarity of scans
            - it is likely that careful, gentle catheterisation can be carefully attempted with little evidence of extension of urethral lacerations or increased haemorrhage
    - management
        - urology review
        - may require initial suprapubic catheterisation
        - operative repair
- bladder injury
    - graded as I to V
    - more practically categorised as:
        - contusion or haematoma
        - intraperitoneal rupture
        - extraperitoneal rupture
    - recognition
        - usually associated with pelvic fracture
            - further investigation is essential if there is macroscopic haematuria in a patient with a pelvic fracture
        - penetrating trauma close to the bladder
        - classic triad:
            - haematuria
            - suprapubic pain
            - inability to void
        - pregnant women and intoxicated patients with full bladders are at higher risk
        - investigation involves retrograde cystogram or CT cystography
            - contrast is injected via urethral catheter
            - should be delayed until life-threatening injuries are excluded or stabilised
    - management

        - urology review
        - bladder contusion and haematomas can be observed
        - intraperitoneal rupture requires laparotomy and surgical repair
        - extraperitoneal rupture can often be managed with simple catheterisation (usually about 10 days)
- penile fracture
    - rupture of the corpus cavernosum due to a tear in the tunica albuginea
    - recognition
        - usually the result of vigorous uncoordinated sexual intercourse but can occur from fall and direct trauma
        - suggestive history – sudden detumescence of a previously erect penis following a loud 'crack'
        - penile haematoma, difficulty voiding
        - urethral and corpus spongiosum injury (5-20%) suggested by:
            - blood at the meatus
            - inability to pass urine
            - extravasation of urine
    - management
        - urology review
        - surgical repair
- penile amputation
    - management
        - urology review
        - apply direct pressure to bleeding stump, avoiding tourniquet
        - analgesia
        - wrap amputated part in dry sterile gauze and place in ice (avoid direct contact of the amputated part with ice)
        - surgical reimplantation (ideally <6 hours warm ischaemic time) or reconstruction
- scrotal injury
    - main concern is testicular rupture
    - recognition
        - blunt trauma to the scrotum such as a kick or fall
        - scrotal haematoma or tenderness
        - scrotal ultrasound is the investigation of choice
    - management
        - urology review
        - reduce a dislocated (luxed) testicle in the ED (replace it into the scrotal sac)
        - surgical repair for testicular rupture, haematocele, non-reducible testicular dislocation and scrotal degloving

UP3 urinary retention

- background
    - acute urinary retention is responsible for over 30,000 admissions per year in the UK and many more ED attendances
    - the majority are in men

        - 10% of men have an episode of acute urinary retention between age70 and 75
- pathophysiology
    - four mechanisms of acute urinary retention
        - increased resistance to flow
            - at the bladder neck smooth muscle, resulting in dynamic outflow obstruction
            - mechanical obstruction such as urethral stricture or prostatic enlargement
        - inappropriate detrusor muscle innervation
            - results from neurological causes such as stroke, spinal cord lesions or diabetic neuropathy
        - bladder overdistension
            - e.g. from post-operative pain or alcohol
            - inability to void leads to bladder distension
        - drugs
            - anti-muscarinic and alpha-adrenergic medications
    - the prostate is rich in alpha adrenergic receptors
        - in acute urinary retention, the maximal pressure rise is in the prostatic urethra, the bladder neck is not tightly closed and the tone of the external sphincter is inhibited
        - 5 alpha reductase drugs such as finasteride decrease all symptoms of benign prostatic hypertrophy and therefore the incidence of acute urinary retention when taken long term
            - frustrating side effects such as impotence and decreased libido occur
    - it is important to always consider neurological causes for acute urinary retention
- drug causes of acute urinary retention
    - decongestants
        - phenylephrine
        - pseudoephedrine
    - antihistamines
        - diphenhydramine
        - Phenergan
    - amphetamines
    - morphine
    - beta agonists
        - atropine
        - isoprenaline
    - hyoscine
    - tricyclic antidepressants
    - nifedipine
- aetiology of acute urinary retention
    - anatomical
        - urethral stricture
        - BPH
        - acute prostatic haematoma

        - prostate cancer
        - bladder neck contracture
        - urethral stone
        - foreign body
        - iatrogenic – e.g. urinary stent occlusion
    - functional
        - neurogenic
            - neurogenic bladder
        - neurological disease
            - MS
            - Parkinson's
            - DESD (detrusor – external sphincter dyssynergia – caused by neurological lesions between brainstem and spinal cord)
            - tabes
            - Alzheimer's
        - spinal cord injury
        - CVA
        - tumour
        - AP resection
        - spinal anaesthesia
        - lower tract instrumentation
        - traumatic pain
        - alcohol toxicity
        - UTI
        - acute prostatitis, E coli, proteus
    - female specific causes
        - organ prolapse
        - pelvic mass
        - gravid uterus
        - vulvovaginitis
        - herpes
- clinical assessment
    - history
        - often elicits progression of benign prostatic hypertrophy
            - gradual deterioration of lower urinary symptoms such as urge, hesitancy, frequency and post micturition dribbling
        - full drug history including recreational drugs
        - screening for constipation, UTI, alcohol excess
        - associated urinary symptoms including haematuria
        - direct questioning for neurological symptoms
        - direct enquiry about urogenital conditions such as herpes that can cause retention through discomfort
        - causes such as infection and constipation should be treated before trial without catheter
    - examination
        - distended bladder arising from the pelvis
            - cannot get below it like other abdominal masses

                - usually midline and dull to percussion
                - occasionally huge bladders can rise above the umbilicus
            - careful inspection of external genitalia for phimosis, trauma or blood in males and vesicles in females
            - digital rectal examination
                - mandatory to assess the prostate and sphincter tone
                - perianal sensation and tone must be documented
                - should be performed after catheterisation
                - size, consistency and contour of prostate gland should be documented
            - bladder scan
- investigation
    - U&Es
    - FBC
    - urine sample for microscopy
    - US renal tract if any renal impairment present
    - if PSA is to be tested it should be deferred by two weeks
- management
    - catheterisation
        - usually urethral
        - occasionally suprapubic
            - US guidance preferred
            - unguided suprapubic catheters are contraindicated in:
                - past history of bladder cancer
                - history of haematuria
                - suspicion of clot retention
                - (they are red flags for bladder cancer and need to avoid seeding along the catheter track)
        - documentation:
            - post-catheterisation residual volume
            - features of prostate examination
            - renal function
            - catheter type and gauge
            - ease of introduction
            - consent
    - post-obstructive diuresis
        - some patients can pass 8-20 litres/day
        - cardiac failure or renal insufficiency patients (particularly if they have marked peripheral oedema) are at high risk
        - severe dehydration and postural hypotension can occur
        - hourly urine outputs should be <200ml as a rule
        - high risk patients often have a residual volume >1 litre and impaired renal function
    - up to 70% of men will have recurrent retention within one week if the bladder is simply drained
    - evidence suggests that suprapubic catheters offer easier TWOC and lower rate of UTIs, with fewer urethral strictures

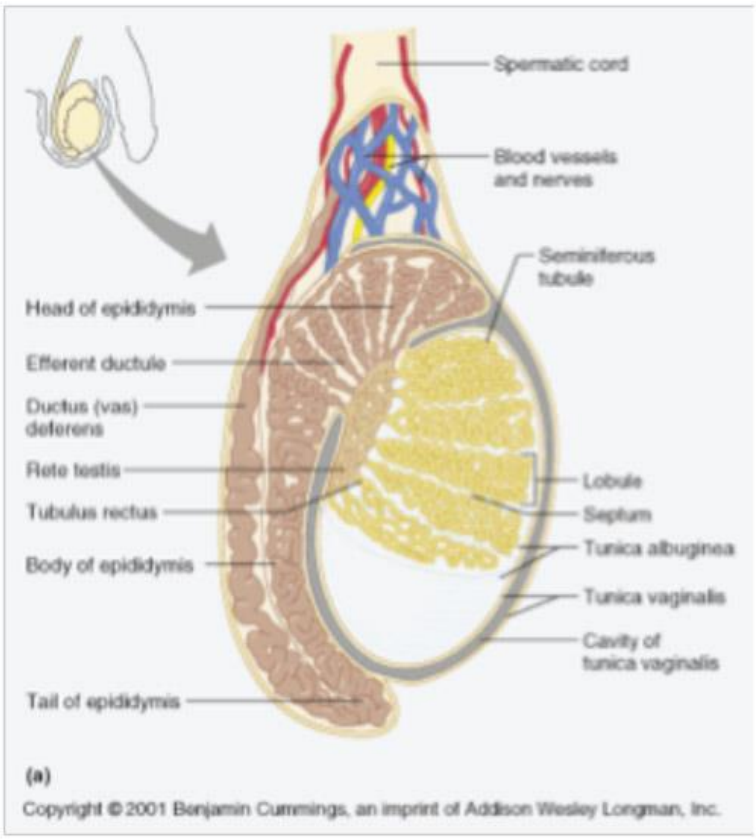
- follow up
    - retention secondary to constipation, UTI with no previous urinary tract symptoms or postoperative pain does not require follow up
        - TWOC may be attempted after treatment
    - early TWOC may be considered for patients with a residual volume of less than 1 litre
    - referral to a urology clinic with catheter in situ is advised for most
        - urology referral is appropriate for most cases as they are likely to require surgical intervention in the future
    - alpha blockers may be prescribed for symptoms of BPH if local policy recommends it
    - contraindications to discharge with a catheter:
        - renal impairment
        - significant post-obstruction diuresis
        - sepsis
        - dehydration
        - inability to manage at home
- suprapubic catheterisation
    - uses the Bonanno technique (small calibre catheter with needle for puncture within the lumen) or the Bard trocar model
    - techniques are similar
        - ensure adequate filling of the bladder, confirm by ultrasound and use direct puncture behind the pubic bone angled downwards with the Bonnaro kit or skin nick and trocar with the Bard method
        - local anaesthesia is essential in both cases
- removal of a non-deflating catheter
    - options include:
        - cutting the catheter
            - only works when the valve flap that retains fluid is in the external segment
        - overinflation
            - requires up to 200ml of fluid
            - allows leaves retained balloon pieces that need to be removed endoscopically
                - otherwise will act as a nidus for infection or stone formation
    - retention of catheter fragments are also common if corrosive substances such as ether or acetone are used
        - they can cause a chemical cystitis and should not be used
    - ultrasound guidance may be used to rupture the balloon transabdominally
    - sliding a central wire guideline down the balloon channel to release the retaining valve can also be effective
    - in all cases the balloon must be inspected to determine whether cystoscopy is needed to remove fragments
- prostate examination
    - the prostate is a heart-shaped organ with the apex located distally
    - prostate examination may raise PSA and contaminate mid stream urine collection
    - try to distinguish gland tenderness from normal discomfort of the examination

    - a normal prostate feels like the end of a nose, and a cancerous prostate feels like the bony areas of a chin
    - most cancer arises in the lateral areas
    - severity of hypertrophy is more accurately determined by severity of symptoms and size of residual volume than by size on rectal examination
- common problems
    - if there is phimosis and the opening is adequate, try passing the catheter blind
        - if the opening is too narrow, try dilating with sounds or using a smaller catheter
        - if in doubt seek expert help
    - if the catheter will not pass the prostate, try a catheter with a larger diameter
        - the urethra is not narrow but is pushed flat by the prostate – a larger catheter pushes the prostate lobes to the sides to allow passage
        - another option is a silicone catheter (more rigid)
    - if the catheter does not pass the bladder neck, try a smaller size
- female acute urinary retention
    - relatively uncommon and often poorly managed
    - underlying abnormality is often detrusor failure rather than outlet obstruction
    - investigations should focus on identifying serious or reversible causes
        - should include detailed history and examination, urine dipstick, culture and pelvic ultrasound
            - pelvic examination and neurological assessment should be included
    - patients with apparently idiopathic retention should be referred to a urologist with a special interest in bladder dysfunction for consideration of urodynamics
- chronic urinary retention
    - symptoms generally gradual in onset and may not be noticed
    - symptoms include:
        - frequency
        - urgency
        - hesitancy
        - poor stream
        - post-micturition dribbling
        - nocturia
        - new onset enuresis
        - urinary incontinence
        - sensation of incomplete voiding
        - ‘double’ or recurrent voiding
        - symptoms consistent with urinary tract infection
        - increasing lower abdominal discomfort
            - may represent acute on chronic retention
        - acute urinary retention
        - lethargy, pruritus, recurrent infections, hypertension due to chronic kidney disease
    - management generally includes:
        - intermittent self-catheterisation if possible, may require indwelling catheter (particularly if impaired renal function or hydronephrosis
        - surgery may be offered in some cases
        - review of medication and stopping any contributing medications

- lifestyle advice
    - regulating fluid intake and avoiding evening drinking
    - reducing alcohol intake
    - reducing tea and coffee intake
- bladder retraining and regular voiding
- drug therapy
    - alpha blockers for BPH

UP4 testicular pain/swelling

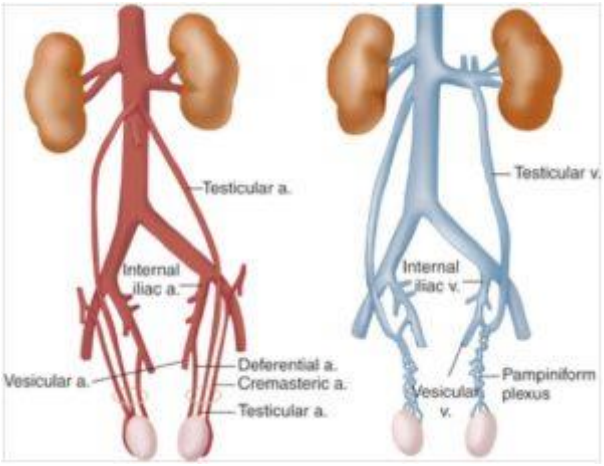
- testicular anatomy



https://www.rcemlearning.co.uk/reference/testicular-pain/#1571735796047-e9857cd1-b43c

- measures 4cm x 3cm x 2.5cm on average
- testis descends during development from the posterior abdominal wall, through the inguinal canal to the scrotum
- nervous, arterial, venous and lymphatic system travel via the spermatic cord
- the testis is suspended in the scrotum by the spermatic cord
- tunica vaginalis
    - derived from abdominal peritoneum
    - separates the testis and epididymis from the scrotal wall
    - covers the exterior anterolateral aspect of the testis, fully surrounds the testis and fixes it to the posterior wall
    - fluid accumulating in the tunica vaginalis leads to hydrocoele (water), haematocoele (blood) or pyocoele (pus)
- tunica albuginea

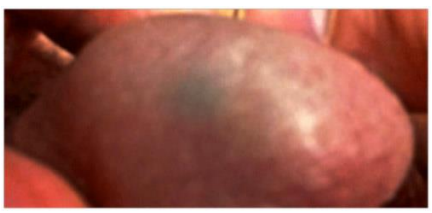
        - dense fibrous fascia enclosing the testis
        - posteriorly penetrates the parenchyma of each testis
        - forms the mediastinum and separates each testis into around 250 lobules
        - each lobule contains 1-4 convoluted seminiferous tubules
    - functions are sperm production and hormone synthesis
        - the basement membrane of the seminiferous tubules contains 2 cell types:
            - Sertoli cells support the structure
            - spermatogenic cells produce sperm
        - the stroma between the cells contains connective tissue which contains Leydig cells
            - responsible for producing testosterone in response to luteinising hormone (LH)
    - spermatozoa, once produced in the seminiferous tubules, collect in the rete testis
        - they then travel via the efferent tubules to the epididymis for storage
        - the epididymis is in the posterolateral aspect of the superior pole of the testis
    - testicular appendages
        - appendix testis and appendix epididymis
        - embryological remnants which serve no purpose but may twist and cause pain
            - common in pre-pubescent boys, does not require surgical intervention if diagnosis is certain
    - blood supply to testes
        - arterial supply is directly from the aorta
            - the gonadal arteries branch off at L2, just below the renal arteries
            - the testicular arteries travel in the spermatic cord with the cremasteric and differential artery, where they anastamose with the vasa deferentia that branch off the internal iliac artery
        - drainage
            - into the pampiniform plexus, a network of veins within the scrotum
                - allows counter current cooling of the testes
                - defective valves or vein compression can cause them to become enlarged, leading to varicocoeles
            - the pampiniform plexus becomes the spermatic vein at the junction of the internal inguinal ring
                - the right spermatic vein drains directly into the vena cava, just below the renal vein
                - the left spermatic veins drains into the left renal vein before it joins the vena cava
    - varicocoeles
        - can cause an aching or dragging sensation within the scrotum
        - because of the drainage of the left testis into the left renal vein, a left renal cancer can present with a left varicocoele
            - left varicocoeles should always be investigated



https://www.rcemlearning.co.uk/reference/testicular-pain/#1571735796047-e9857cd1-b43c

  - nervous supply to the testes
    - nervous supply is extensive, pain in the scrotum may be referred from visceral or somatic structures
    - autonomic nerve supply is from the testicular plexus which originates from the paraaortic ganglia
    - ramus genitalis (branch of the genitofemoral nerve) and the ilioinguinal nerve supply the scrotal skin and muscles in the groin area
  - lymphatic drainage of the testes
    - drains to the lumbar and para-aortic nodes at L1
      - means testicular cancers or infections may not lead to palpable lymphadenopathy
    - lymphatic drainage of the scrotum is to the superficial inguinal nodes
- common causes of acute testicular pain
  - testicular torsion
  - epididymo-orchitis
  - torted appendages
- other causes include:
  - trauma
    - can cause torsion, testicular rupture or haematoma
    - needs referral to urology
  - inguinal hernias
    - may have a history of fluctuant testicular swelling
    - have the potential to become irreducible and incarcerated
  - Fournier's gangrene
    - necrotising fasciitis of the perineum
    - more common in diabetics and vasculopaths
    - presents with severe pain and rapidly spreading necrotic cellulitis
  - malignancy
    - testicular cancer typically presents in men aged 15-40
      - small hard lump within the testis, with or without pain
      - US indicated if any concerns
  - oedema/swelling
    - peripheral oedema can progress up to the testes and cause diffuse swelling and pain

        - requires diuretics and medical management
    - cysts/hydrocoeles
        - should be relatively painless but may cause dragging or heavy sensation
    - referred pain
        - e.g. renal colic, AAA
- history
    - age
        - torsion less likely in elderly but can occur at any age
    - pain
        - sudden onset of pain in 90% of patients with testicular torsion
        - gradual onset more likely with torsion of an appendage but may represent intermittent torsion
        - previous torsion and orchidopexy makes torsion highly unlikely
    - sexual history
        - sexually transmitted infections are a common cause of epididymo-orchitis
        - risk factors include:
            - previous STI
            - urethral discharge
            - number of sexual partners in past 12 months
            - men who have sex with men
- examination
    - abdomen
        - tenderness
        - hernias
        - lymphadenopathy
    - scrotum and penis
        - inspection
            - swelling
            - erythema
            - lie of the testicle
                - bell clapper deformity leads to horizontal lie of the testicle and increased free space for the testicle to move
                - increases risk of torsion
                - deformity thought to be present in 10% of the population but torsion does not occur in all cases
            - penile rash
            - discharge
            - blue dot sign
                - occurs in a torted testicular appendage, can be difficult to see



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- palpation
    - tenderness or masses in testicle or scrotum
        - rapid localised oedema in torsion can make assessment difficult
        - tenderness in torsion is usually diffuse, in epididymitis it is localised within the testis
    - lie of the testis
        - normally vertical with the epididymis above the superior pole in a posterolateral position
        - a torted testis is often high riding with a horizontal lie
    - cremasteric reflex
        - elicited by stroking the inner thigh
            - elevation of the ipsilateral testis should occur
            - commonly absent in testicular torsion
    - Prehn's sign
        - elevation of the scrotum
        - in theory relieves the pain of epididymitis but not torsion

UP5 loin pain

- background
    - commonly originates from the kidneys
    - epidemiological considerations include:
        - renal stones are common and more common with a positive family history
        - first presentation of renal stones in the elderly or children is uncommon (but should be considered)
        - renal stones are more common in men
        - pyelonephritis and urinary infections are more common in women
        - abdominal aortic aneurysms are common in elderly patients with risk factors
        - back pain is very common in all age groups
- history
    - when pain started
    - sudden or gradual onset
    - continuous or intermittent pain
    - nature of pain (e.g. stabbing, burning, gripping)
    - any nausea or vomiting
    - aggravating or relieving factors
    - effect of movement and deep inspiration
    - any radiation of pain
    - any urinary symptoms and whether the urine looks normal
    - pyrexia and/or rigors
    - any similar pain before
    - past medical history
    - medication
    - brief family history
    - risk factors for causes in the differential where appropriate
- examination
    - pyrexia
    - dyspnoea

- shock
- patient lying still (acute abdomen) or writhing around (colic)
- temperature, pulse and blood pressure
- respiratory and abdominal examination
- back examination

- differential
  - renal causes
    - renal colic
      - sudden onset
      - very severe
      - patient may writhe around
      - radiation to groin and anteriorly
      - tenderness over renal angle
      - more constant and persistent than biliary colic
      - gross or microscopic haematuria in 85%
      - a stone that is moving may be more painful than one that is static
    - pyelonephritis
      - high temperature
      - ill patient
      - rigors
      - urinary tract infection symptoms
      - vomiting
      - dull ache
      - leucocytosis
      - pyuria
    - blood clots
      - sudden ureteric obstruction and colic
      - causes include:
        - renal biopsy
        - bleeding disorders
        - tumours of the kidney or renal pelvis
        - haemophilia
        - sickle cell disease
        - glomerulonephritis
      - an uncommon cause is idiopathic loin pain haematuria syndrome (loin pain, haematuria and no apparent cause)
    - papillary necrosis
      - acute obstruction of the ureter from the sloughed papilla
      - women affected more than men
      - causes include:
        - analgesic abuse
        - cirrhosis
        - recurrent pyelonephritis
        - urinary tract obstruction
        - tuberculosis
        - sickle cell disease
        - renal transplant rejection

        - diabetes
    - kidney tumours
        - gradual onset of pain
        - may be painless haematuria and/or mass
        - commonest forms are clear cell carcinoma in adults and Wilms' tumour in children
    - pelvi-ureteric junction obstruction
        - usually follows drinking large amounts of fluid
            - diuresis causes distension of the renal pelvis and colic
    - renal infarction
        - frequently misdiagnosed (e.g. as acute renal colic, pyelonephritis or acute abdomen)
        - acute renal infarction affects both kidneys and both sexes equally
        - average age 65
        - risk factors include age and risks for thromboembolism
        - unilateral flank pain and haematuria
        - other common symptoms include fever, nausea and vomiting
        - LDH and WCC are usually elevated
        - diagnosis is usually by CT
        - treatment is by IV heparin and/or intra-arterial thrombolysis
- other local causes
    - dissecting abdominal aortic aneurysm
        - can cause loin pain similar to renal colic
        - aneurysm can be adjacent to the ureter and cause haematuria from irritation or trauma
        - consider in older patients presenting with apparent renal colic for the first time
    - herpes zoster
        - burning pain in a band corresponding to a dermatome
        - pain may precede the rash
        - diagnosis is easy when the rash appears
        - skin is tender
    - muscular pain
        - aching discomfort exacerbated by lifting and bending
        - the affected area is likely to be tender
        - there may be a history of injury
    - injury
        - injury to the lower thoracic or lumbar nerve roots
            - causes radiculitis
            - suspect in patients with normal urinalysis and normal urinary tract imaging
        - injury to the 10^{th}, 11^{th} or 12^{th} ribs
            - has the distribution of renal colic but is usually sharp or stabbing
            - often acute and can radiate anteriorly and inferiorly like renal colic
            - movement exacerbates the pain

                - fracture of a transverse process in the thoracic or lumbar vertebrae
            - retroperitoneal fibrosis
                - dull pain, insidious in onset, becoming progressively more severe
                - there is fibrous overgrowth of the retroperitoneal organs from the midline
                - when it involves the ureters it causes hydronephrosis and possibly renal failure
                - usually idiopathic
                    - has been associated with use of methysergide for migraine and some malignancies
                - males are affected twice as commonly than females
                - usually aged 40-60 years
                - pain is usually located first in the lower flank and abdominal regions
                    - testicular pain and periumbilical pain develop later
    - pulmonary causes
        - a sharp, stabbing pain can originate from the lung or pleura
        - pain is typically pleuritic
        - possible causes include tuberculosis, pneumonia and pulmonary embolism
    - less common causes
        - Crohn's disease
        - diverticulitis
        - appendicitis
        - myocardial infarction
        - renal vein thrombosis
        - glomerulonephritis
        - Berger's disease
        - acute nephritis
        - polycystic kidney disease
        - cholecystitis
        - adrenal tumours and haemorrhage
        - colon cancer
        - splenic infarction
- investigations
    - urine
        - microscopy
            - haematuria with renal stones
            - pyuria with infection
            - casts in kidney disease
        - urine pH <6.0 can lead to suspicion of uric acid stone
            - if pH >8.0, infection with a urea-splitting organism such as Proteus, Pseudomonas or Klebsiella is likely
        - urinary crystals of calcium oxalate, uric acid, cystine
        - mild leucocytosis is common in renal colic; WCC >15,000 suggests infection
- imaging
    - CTKUB
        - can show stones and sometimes other pathology
    - x-ray KUB

        - may show abnormal shadows of the renal tract
        - 70% of renal stones are radio-opaque
        - phleboliths can be mistaken for stones
    - ultrasound
        - shows stones if >0.5cm in diameter
        - shows hydronephrosis and the size and shape of the kidneys
    - further urological studies
        - may include IV pyelogram or retrograde pyelogram
- management
    - depends on diagnosis
        - stones
            - analgesia
            - oral fluids
            - most 2-4mm stones pass spontaneously
            - stones >7mm are unlikely to pass spontaneously
        - obstruction with infection
            - likely to require percutaneous nephrostomy

UP6 haematuria

- background
    - blood may originate from the kidney or collecting system
    - significant haematuria is described as:
        - any single episode of visible haematuria
        - any single episode of symptomatic microscopic haematuria in the absence of urinary tract infection or other transient causes
        - persistent asymptomatic microscopic haematuria in the absence of UTI or other transient cause
            - persistence defined as 2 out of 3 positive dipsticks
- aetiology
    - common causes include UTI, bladder tumours, urinary tract stones, urethritis, benign prostatic hypertrophy, prostate cancer
    - up to 5% of patients with microscopic haematuria are found to have a urinary tract malignancy
    - infection
        - cystitis
        - tuberculosis
        - urethritis
        - schistosomiasis
        - infective endocarditis
    - tumour
        - renal carcinoma
        - Wilm's tumour
        - carcinoma of the bladder
        - prostate cancer
        - urethral cancer
        - endometrial cancer
    - trauma

        - renal tract trauma
            - accidents
            - catheter
            - foreign body
            - prolonged severe exercise
                - causes blunt trauma of the posterior bladder wall against the trigone
                - should settle over 48-72 hours
            - rapid emptying of overdistended bladder
    - inflammation
        - glomerulonephritis
        - Henoch-Schönlein purpura
        - IgA nephropathy
        - Goodpasture's syndrome
        - polyarteritis
        - post-irradiation
    - structural
        - calculi
            - renal
            - bladder
            - ureteric
        - simple cysts
        - polycystic kidney disease
        - congenital vascular anomalies
    - haematological
        - sickle cell disease
        - coagulation disorders
        - anticoagulation therapy
    - surgery
        - invasive procedures to the prostate or bladder
    - toxins
        - sulfonamides
        - cyclophosphamide
        - NSAIDs
    - others
        - genital bleeding
            - including child abuse
        - menstruation
        - fabricated or induced illness
- assessment
    - full urological history
    - palpation of abdomen
    - blood pressure
    - features suggesting a renal cause include:
        - hypertension
        - altered renal function tests
        - proteinuria

        - known previous renal problems
        - renal mass
        - glomerular red cells in urine (red cells with irregular contours and shape)
    - haematuria without proteinuria does not necessarily indicate a non-glomerular origin
        - glomerular bleeding is not necessarily accompanied by proteinuria
    - risk factors for urological cancer
        - age >40
        - smoking
        - exposure to occupational chemicals
            - benzenes
            - aniline dyes
            - aromatic amines
        - cyclophosphamide
        - analgesic abuse
        - prior pelvic irradiation
        - recurrent UTI
        - schistosomiasis
    - timing in relation to the stream
        - initial haematuria suggests a urethral source, terminal haematuria suggests a bladder neck or prostatic urethral source
        - haematuria through the stream suggests bladder or upper tract pathology
        - haematuria between voiding, noticed as staining in the underwear, while voided urine is clear, suggests lesions at the distal urethra or the meatus
    - systemic symptoms
        - recent weight loss
        - upper respiratory tract symptoms and possible streptococcal sore throat
            - consider post streptococcal glomerulonephritis and acute nephritic syndrome
        - gastrointestinal symptoms may be part of a haemolytic uraemic syndrome
        - atrial fibrillation can cause renal emboli with haematuria
        - sexual history, including recent coitus – haematuria is common post-coitally
        - haematuria associated with menstruation and associated with dysmenorrhoea may indicate endometriosis
    - drug history
        - NSAIDs
        - penicillin
        - cephalosporins
        - cyclophosphamide
        - anticoagulants
    - physical examination
        - palpable bladder
        - abdominal masses
        - rectal masses
        - pelvic masses
        - inspection of the vagina and cervix
            - vaginal bleeding must be excluded, by speculum examination if needed

        - examination of the external male genitalia
- differential for dark red urine
    - haemoglobinuria
        - dipstick positive but no red cells on microscopy
    - myoglobinuria
    - food, e.g. beetroot
    - drugs
        - rifampicin
        - nitrofurantoin
        - senna
    - porphyria
        - urine darkens on standing
    - bilirubinuria
        - obstructive biliary disease
- other colours of urine
    - dark yellow
        - drugs
            - chloroquine
            - sulphonamides
        - bilirubin
        - foods
            - carrots
            - riboflavin
            - vitamin A
    - orange
        - drugs
            - phenolphthalein laxatives
            - rifampicin
            - sulphasalazine
    - blue-green
        - pseudomonas
        - drugs
            - amitriptyline
            - NSAIDs
            - promethazine
    - red-brown
        - sorbitol
        - porphyria
        - foods
            - rhubarb
            - fava beans
- investigation
    - urine dipstick
        - no correlation between the extent of haematuria and the extent and severity of underlying disease
        - up to 4% of patients with normal dipsticks have abnormal microscopy
        - leucocyte esterase

- detects the presence of white blood cells as a consequence of infection or inflammation
- a negative dipstick result does not exclude a UTI
- false positive readings are less common than false negatives
- other causes of sterile pyuria include:
  - stones
  - tubulointerstitial nephritis
  - papillary necrosis
  - TB
  - interstitial nephritis
- white cells can persist in the urine for some time after treatment so treatment should not be started blindly

- nitrites
  - nitrates are converted to nitrites in the bladder following a period of incubation with organisms
  - most reliable when used on the first void of the day
  - positive predictive value of 96% for urinary tract infection but low negative predictive value
- blood
  - does not differentiate between haemoglobin from red cells and myoglobin from breakdown of skeletal muscle
  - total obstruction may initially have no red cells present

- urine culture
  - not routinely needed in young women with typical symptoms and positive nitrites and leucocytes
  - culture should be done in all other cases, especially males, the immunocompromised, pregnant women and those with UGT obstruction and treatment failures
  - delays in transporting samples to the lab can result in false positives so samples should be transported and refrigerated promptly
- CT
- endoscopy
- follow up required for all cases without a clear cause which resolves with managment

UC1 epididymo-orchitis

- pathophysiology
  - clinical syndrome consisting of pain, swelling and inflammation of the epididymis +/- testis
  - most common cause is local extension of infection from the urethra (sexually transmitted) or bladder (urinary tract pathogens)
  - causes:
    - usually sexually transmitted such as Chlamydia trachomatis or Neisseria gonorrhoea in under 35s
    - usually urinary pathogens in over 35s
      - risks include recent instrumentation or catheterisation
    - can be enteric organisms in men who have penetrative anal intercourse

        - other causes to consider include:
            - mumps
            - tuberculosis
            - amiodarone treatment
            - fungi
- clinical features
    - symptoms
        - unilateral scrotal pain and swelling, relatively acute
        - may have urethritis or urethral discharge in sexually transmitted cases
        - symptoms of mumps
            - headache, fever, parotid swelling
        - systemic symptoms of tuberculosis
    - signs
        - tenderness to palpation on affected side
        - palpable swelling of the epididymis starting with the tail at the lower pole of the testis, spreading towards the head at the upper pole of the testis +/- involvement of the testicle
    - other possible signs:
        - urethral discharge
        - secondary hydrocoele
        - erythema and/or oedema of the scrotum on the affected side
        - pyrexia
    - if any concerns about possible torsion, urgent review required
- complications
    - reactive hydrocoele
    - abscess formation and infarction of the testicle (rare)
    - infertility
        - poorly understood pathophysiology
- investigations
    - first pass urine for microscopy and culture
    - mid stream urine for microscopy and culture
    - urine dip for evidence of UTI
- management
    - general advice
        - rest, analgesia (NSAIDs), scrotal support
        - abstinence from sexual intercourse until they and their partner(s) have completed treatment and follow up
        - explanation of condition with emphasis on long term implications, with clear and accurate written information
    - treatment
        - should be empirical whilst awaiting results
        - if likely sexually transmitted:
            - ceftriaxone 500mg IM single dose plus doxycycline 100mg PO BD for 10-14 days
        - if likely due to enteric organisms:

                - ofloxacin 200mg PO BD for 14 days
                - OR ciprofloxacin 500mg PO BD for 10 days
        - consider inpatient management for IV fluids and antibiotics if severe or with features of bacteraemia
        - notification and treatment of sexual partners required (refer patient to GUM clinic)
- follow up
    - should be reviewed after 3 days if no improvement for re-evaluation
    - further follow up at 2 weeks to assess compliance with treatment, partner notification and improvement of symptoms
    - test of cure if treated for gonorrhoea
    - ultrasound scan/surgical assessment if no significant improvement in swelling/tenderness

UC2 renal stone disease

- pathophysiology
    - pain
        - due to ureteric muscle contracting in an attempt to move the stone
        - caused by obstruction of flow in the ureter
        - prostaglandin synthesis is increased with resultant vasodilatation causing diuresis
            - this further increases pressure
            - prostaglandins can also cause smooth muscle spasm in the ureter
    - stone composition
        - predominantly calcium oxalate and/or phosphate (60-80%)
        - struvite (10-15%)
        - cystine (1%)
        - uric acid (1%)
        - others
    - most common factor in development is low urine volume
        - output of 3 litres/day should be encouraged
    - a causative factor is not normally identified and investigation is probably not required for first presentations
    - risks:
        - lifetime risk is 10%
            - recurrence rate 50% over 10 years
        - peak age 30 years
        - twice as common in men
        - three times more common where there is a family history
        - more common in Caucasians than Asian or Black people
    - biochemical predisposition:
        - hypercalciuria
        - hyperuricosuria
        - hyperoxaluria
        - hypocitraturia
        - cystinuria
        - urease producing organisms

        - urea splitting organisms (proteus, klebsiella, serratia, mycoplasma) – produce struvite stones
    - anatomical predisposition:
        - pelvi-ureteric junction obstruction
        - hydronephrotic renal pelvis or calyces
        - calyceal diverticulum
        - horseshoe kidney
        - ureterocoele
        - vesicoureteric reflux
        - ureteral stricture
        - medullary sponge kidney
    - other aetiologies:
        - insulin resistant states
        - hypertension
        - primary hyperparathyroidism
        - gout
        - metabolic acidosis
- presentation
    - unilateral loin to groin or renal pain
        - colicky
        - radiates to scrotum/tip of penis or labia majora
    - writhing or restless
    - nausea
    - larger stones can present with haematuria, infection, decreased renal function
    - pyuria can be present, usually from ureteral irritation rather than infection
- differentials
    - pancreatitis
    - appendicitis
    - ovarian pathology and torsion
    - pelvic inflammatory disease
    - pregnancy
    - renal infarct
    - aortic aneurysm
    - cholecystitis
    - renal carcinoma, pyelonephritis
    - incarcerated hernia
    - diverticular disease
    - pneumonia
- investigations
    - urinalysis
        - microscopic haematuria in 90% of cases (but absent in 10%)
        - send for microscopy and culture if concerns about infection
    - 24 hour urine collection for recurrent stone formers:
        - urine volume
        - calcium
        - oxalate
        - uric acid
        - citrate

        - sodium
        - creatinine
    - blood tests:
        - FBC, U&E
        - consider amylase, phosphate, urate, bicarbonate, calcium
    - abdominal radiography
        - KUB view
        - low sensitivity for detecting stones (40-50%)
            - only 59% of stones are visible on x-ray
        - not sensitive for non-radio-opaque stones or for other causes of ureteric obstruction
        - specificity of 77% for renal stones
            - due to pelvic calcifications and phleboliths
        - can be used in follow up of known stones
    - intravenous urography
        - stones visualised as lucent filling defects in contrast
        - standard films are plain KUB, 20 minute film and post micturition film
        - delayed films can demonstrate obstruction
        - can be better than CT at assessing degree of obstruction
        - CT generally considered superior
        - avoid in:
            - renal failure
            - contrast allergy
            - pregnancy
            - diabetics on metformin
        - disadvantages of IVU
            - difficult to interpret
            - needs contrast administration
            - radiation (less than CT)
            - delay in obtaining relevant information about possible site of obstruction
    - CTKUB
        - modality of choice
            - rapid
            - can measure size of calculi
            - assesses degree of obstruction
        - false negative rate is 2-7%
        - secondary signs such as hydronephrosis, perinephric oedema and stranding and periuretal oedema are often seen
            - perinephric stranding appears at 2 hours and is maximal at 8 hours
        - over 99% of stones are seen on CT
        - the majority of alternative diagnoses are also detected by CT, such as pyelonephritis, renal mass, adnexal pathology, diverticulitis, appendicitis
    - ultrasound
        - lower sensitivity (24-77%) compared to CT (96%)
        - specificity 100% if both US and CT used
        - should be first line in pregnancy and children

        - visualisation of ureteral jets within the bladder lumen disappears with obstruction
- management
    - 90% of small stones (≤5mm) pass spontaneously
    - on average, spontaneous passage of stones of all sizes is:
        - 25% in the proximal ureter
        - 45% in the mid ureter
        - 70% in the distal ureter
    - urologists often advise sieving urine to retrieve stone fragments for analysis
    - analgesia
        - rescue medication requirements are less with NSAIDS
        - lower pain scores are reported with NSAIDS
        - higher rates of adverse effects are seen with opiates
        - NSAIDS may reduce ureteric inflammation and facilitate stone passage
    - nephrostomy
        - required urgently in septic patients with hydronephrosis
    - stenting
        - may be required for stones causing significant obstruction and pain pending definitive treatment
        - on x-ray proximal coil is in the renal pelvis and distal coil in the bladder
        - nephrostomy required in the 20% of cases where stenting fails
        - stents can cause multiple complications:
            - renal failure
            - ureteral stricture
            - urine extravasation
            - perinephric abscess
            - xanthogranulomatous pyelonephritis
                - suppurative granulomatous reaction to chronic infection, often where there is chronic obstruction
                - requires surgery
- indications for admission
    - uncontrolled pain
    - bilateral blockage
    - single kidney
    - anuria
    - signs of infection
- discharge
    - patients who are pain free and apyrexial with no high risk factors or complications
    - must have adequate analgesia
    - must have outpatient urology follow up
        - stone fragments should be kept and taken to urology clinic
    - tamsulosin may be useful to help with stone passage
    - advice:
        - return to the ED if pain returns
        - return immediately if unwell or febrile
        - how to use analgesia
        - advise to increase fluid intake and aim for 2 litres of urine/day

- definitive treatment
    - preferably assistance of spontaneous passage
    - extra-corporeal shockwave lithotripsy (ESWL)
        - used for proximal non-obstructing calculi
        - cannot be used in pregnancy, coagulopathy, uncontrolled hypertension, febrile UTI
    - endoscopic retrieval for large stones, multiple stones or stones causing significant obstruction
        - increased complications to ESWL but also increased stone free rate

UC3 phimosis/paraphimosis

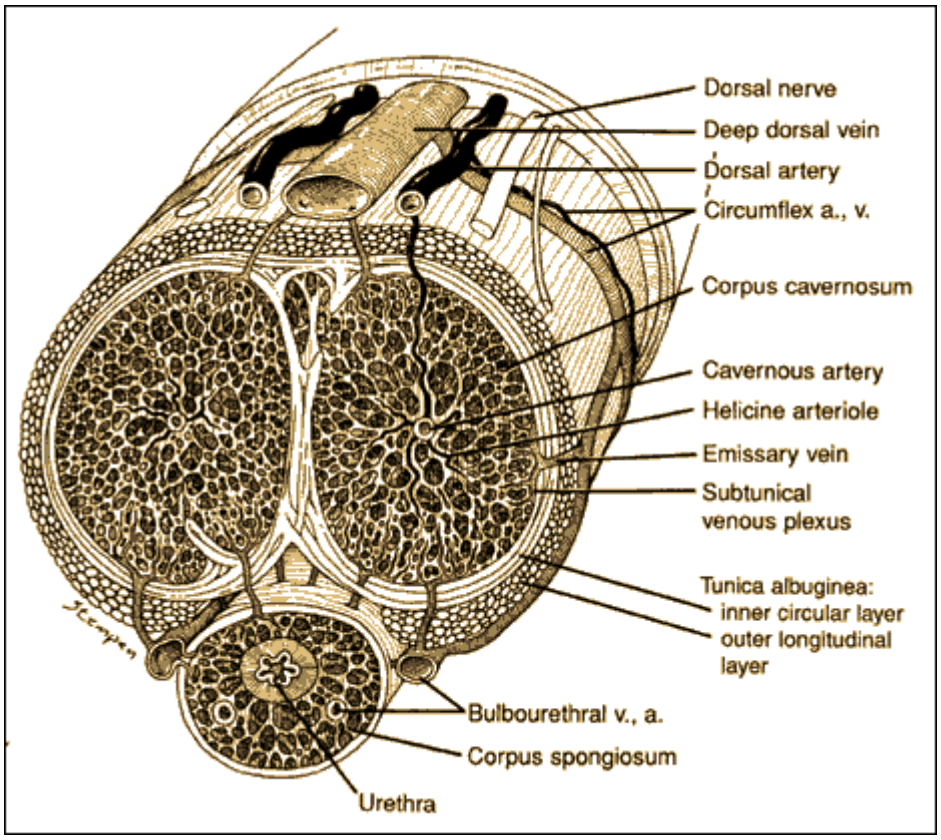
- phimosis
    - physiological
        - almost all boys have a non-retractile foreskin at birth
        - the inner foreskin is attached to the glans
        - foreskin adhesions break down and form smegma pearls (white cysts under the foreskin) which are extruded
        - the foreskin does not retract before the age of 2 years
            - most boys will have a retractile foreskin by 10 years
            - almost all will have a retractile foreskin by 16-17 years
        - phimosis is not a problem unless it causes issues such as urinary obstruction, haematuria or pain
    - pathological
        - the prepuce is tight and unable to be pulled back over the glans
        - risk is increased by poor hygiene and enthusiastic attempts to correct congenital phimosis
        - usually due to episodes of foreskin infection (balanoposthitis)
        - repeated infections lead to scarring, which then leads to further infections
        - usually occurs in uncircumcised males but can occur after circumcision if excessive skin becomes sclerotic
        - female phimosis can also occur (clitoral phimosis) and can contribute to dyspareunia
    - presentation
        - physiological
            - parental concern about foreskin not retracting
            - ballooning during micturition
            - recurrent balanoposthitis
            - recurrent UTIs
        - pathological
            - painful erections
            - haematuria
            - recurrent UTIs
            - preputial pain
            - weak urinary stream
    - examination
        - there may be swelling, redness and tenderness of the prepuce with purulent discharge

        - there may be adhesions visible between the inner surface of the prepuce and the glans or frenulum
        - the frenulum may be shortened with ventral distortion of the glans
        - in physiological phimosis the meatus is healthy and unscarred
        - in pathological phimosis the meatus may be scarred, with a fibrous white ring around the preputial orifice
    - management
        - reassurance and expectant approach if under 2
        - avoid forcible retraction of congenital phimosis
        - encourage good personal hygiene, with cleaning under a retractable foreskin then reducing it to cover the glans
        - topical steroid application may be considered
        - phimosis after 2 years may need referral, particularly if recurrent balanoprosthitis or UTIs
            - options are plastic surgery (including dorsal incision of the foreskin or release of adhesions) or circumcision
    - complications
        - risk factor for penile carcinoma (possibly due to associated infection)
- paraphimosis
    - pathophysiology
        - occurs when a tight prepuce is retracted and is unable to be replaced as the glans swells
        - urological emergency
    - risk factors
        - failure to reduce foreskin after catheterisation is the most common cause
        - scarring after repeated forcible retraction
        - vigorous sexual activity
        - chronic balanoposthitis
        - penile piercing
    - presentation
        - oedema around a constricting band (which is usually the prepuce)
        - may be pain on erection
        - irritability in infants
        - black or blue glans due to necrosis in later stages
    - management (as soon as possible)
        - manual reduction
            - circumferential manual pressure to distal penis to reduce oedema
            - maintain pressure for several minutes
                - or ask patient to do so
                - may need to compress for 5 to 30 minutes
            - release pressure and advance foreskin over glans
                - use thumbs to push glans towards foreskin and fingers to pull foreskin over glans
        - compressive bandage reduction
            - wrap distal penis with compressive bandage in distal to proximal direction
            - leave for several minutes

        - allows oedema to disperse
    - remove bandage and advance foreskin over glans
- ice packs
    - apply in 3 minute intervals to reduce oedema
        - requires careful monitoring for frostbite
- sugar
    - granulated sugar or 50% dextrose on swab applied to distal penis
    - leave for up to an hour for osmotic effect
- urology referral
    - urgent referral if all measures failed
- Dundee technique
    - local anaesthetic (without adrenaline), penile ring block or sedation
    - clean foreskin
    - use 26 gauge needle to make around 20 puncture holes in the foreskin
    - use gentle manual pressure to express oedema fluid from foreskin
    - advance foreskin over glans
- dorsal slit
    - involves local anaesthetic (without adrenaline), using haemostat to create tract between dorsal penis and foreskin, crushing the foreskin along the tract for 10 minutes then cutting the crushed tissue +/- suturing oozing edges

- follow up
    - patients should be referred to urology for consideration of circumcision

UC4 priapism



https://www.rcemlearning.co.uk/foamed/male-urological-issues-in-the-ed/definition

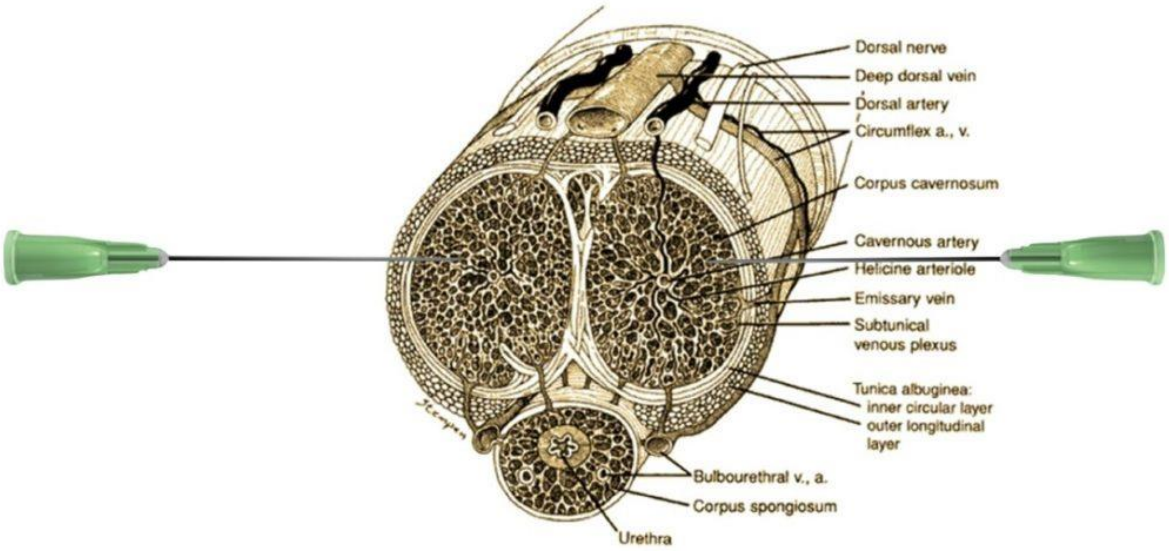
- prolonged, pathological erection of the penis for >4 hours in the absence of sexual desire

- types
  - low flow (ischaemic)
    - most common type
    - veno-occlusion causes pooling of deoxygenated blood in the cavernous tissue
    - rigidity of corpora cavernosa and little or no cavernous arterial flow
    - penile equivalent of compartment syndrome
      - urological emergency
    - painful
  - high flow (non-ischaemic)
    - rare
    - associated with trauma or instrumentation
    - often caused by a fistula between cavernous artery and venous drainage
    - increased arterial flow
    - usually painless
    - usually self resolves
  - stuttering (recurrent or intermittent)
    - repetitive episodes of painful prolonged erection followed by flaccidity
    - priapic episodes are low flow (ischaemic) and can sometimes progress to a major ischaemic episode
- complications

    - penile fibrosis
    - urinary retention
    - incontinence
    - thrombosis and ischaemia
- differential
    - normal sexual arousal
    - penile trauma
    - urethral foreign bodies
    - spinal cord injury
    - Peyronie's disease
    - penile implant
- causes
    - haematological
        - sickle cell anaemia
        - leukaemia
        - thalassaemia
        - myeloma
    - trauma
        - spinal
        - penile/straddle
        - pelvic
    - spider bites
        - Latrodectus envenomation (black widow)
    - medication
        - anticoagulants
        - antihypertensives
        - antidepressants
        - intercavernosal injections
        - erectile dysfunction medications (sildenafil)
    - drugs of abuse
        - cocaine
    - infection
- history
    - duration of erection
    - presence and degree of pain
    - previous episodes of priapism and method of treatment
    - current erectile function
        - use of any therapies
    - prescription and non-prescription medications and recreational drugs
    - sickle cell disease or haemoglobinopathies
    - hypercoagulable states
    - trauma to pelvis, perineum or penis
    - spinal trauma
- clinical features
    - low flow
        - pain
        - rigid penile shaft (corpus cavernosum)
        - flaccid penile glans and spongiosum

- high flow
    - painless
    - partially rigid shaft
    - rigid penile glans

- management (low flow)
    - analgesia
    - may resolve with squats or running in place
    - warm compress
    - consider oral terbutaline 5-10mg
        - beta 2 agonist, increases venous outflow
    - aspiration of blood to determine whether ischaemic or arterial



https://www.rcemlearning.co.uk/foamed/male-urological-issues-in-the-ed/

- Entonox or Penthrox
- clean skin
- use a blue needle to raise bleb of lidocaine at 3 and 9 o'clock or 2 and 10 o'clock positions
- insert green needles at 3 and 9 o'clock, preferably attached to an octopus extension
    - there is a 'give' as it goes through the fibrous Buck's fascia
    - stop once a flash back is achieved
        - avoids causing damage to the central arteriole, creating an iatrogenic fistula
- aspirate 20ml corporal blood on each side
- analyse blood gas sample
    - high CO_2 and acidosis suggests ischaemic priapism
- inject medication
    - usually phenylephrine 200 microgram every 3-5 minutes
        - contraindicated in cardiovascular or cerebrovascular disease, may cause hypertension
        - maximum of 1mg in one hour
    - adrenaline 2ml of 1:100,000

        - up to 5 times in 20 minutes
    - methylene blue
        - 50-100mg intracavernosal injection followed by aspiration and compression
  - urgent theatre transfer if any method unsuccessful
  - wrap penis in elastic bandage if successful
  - some departments may try hydration and exercise for sickle cell patients first
    - give supplementary oxygen
  - there is a risk of impotence with or without intervention – some departments may ask urology to perform the procedure
- management (high flow)
  - analgesia
  - exercise
  - warm compress
  - urology referral for observation
- patients may be suitable for discharge if resolved, with urology follow up

UC5 testicular torsion

- definition
  - twisting of the spermatic cord causes loss of blood supply to the testicle and leads to ischaemia
- risk factors
  - undescended testes
  - bell clapper deformity
- clinical features
  - bimodal presentation
    - first peak in first year of life
    - second peak at puberty
  - half occur during sleep
  - abrupt onset of testicular pain
    - associated with nausea and/or vomiting
  - may have had previous similar, self-resolving episodes
  - may present after scrotal trauma with persistent pain
- examination
  - swollen, high-riding testis
  - unilateral tender, firm testicle
  - transverse testicular lie
  - scrotal erythema, oedema and swelling
  - absent cremasteric reflex on affected side
    - 99% sensitivity
- TWIST score (proposed for evaluating torsion in children)
    - testicular swelling (2)
    - hard testicle (2)
    - absent cremasteric reflex (1)
    - nausea or vomiting (1)
    - high-riding testicle (1)
  - PPV 100% when >5 (urgent urology review)

    - NPV 100% when <2 (potential clinical clearance)
    - scores between 2 and 5 may benefit from US however urology review is advised
- management
    - urgent urological review
    - US only for equivocal cases/late presentation
        - shows unilateral absence of blood flow
        - hypoechoic, heterogenous, enlarged testicle
    - manual detorsion (only if urgent urology review not available)
        - temporising measure
        - IV analgesia or cord block (10ml lidocaine directly into cord at external ring)
        - 'open the book' by twisting testicle outward and laterally
            - grasp testicle with thumb and forefinger, rotate 180 degrees in medial to lateral direction
        - repeat rotation 2-3 times until testicle detorted and pain decreases
        - if pain worse after rotation or rotation not successful, attempt to rotate in opposite direction
    - surgical detorsion and fixation
        - salvage rates:
            - <6 hours – 90-100%
            - 6-12 hours – 20-50%
            - >24 hours – 0-10%
                - intermittent torsion/detorsion means some tissue survival possible

UC6 prostatitis

- background
    - inflammation of the prostate gland
    - can be non-bacterial or bacterial
    - chronic prostatitis is more common than acute prostatitis
    - bacterial prostatitis is more common in the under 35s
- bacterial prostatitis
    - usually gram negative organisms
        - E coli, Enterobacter, Serratia, Pseudomonas, Proteus
    - sexually transmitted infections may be a cause
        - e.g. Neisseria gonorrhoeae, Chlamydia trachomatis
    - rarely caused by Mycobacterium tuberculosis
- non-bacterial prostatitis
    - elevated prostatic pressures
    - pelvic floor myalgia
    - emotional disorders
- risk factors
    - sexually transmitted infections
    - urinary tract infections
    - indwelling catheters
    - post sclerotherapy for rectal prolapse
    - following manipulation of the gland (e.g. post-biopsy)
    - increasing age

- classification
    - acute bacterial prostatitis
    - chronic bacterial prostatitis
    - chronic prostatitis
    - asymptomatic inflammation
- history
    - fever, malaise, arthralgia, myalgia
    - urinary frequency, urgency, dysuria, nocturia, hesitancy, incomplete voiding
    - low back pain, low abdominal pain, perineal pain, urethral pain
        - chronic pelvic pain is the most common finding in chronic prostatitis
    - pain on ejaculation, premature ejaculation
    - urethral discharge
- examination
    - fever may be present
    - acute bacterial prostatitis
        - gland may feel nodular, boggy or normal
        - gland may be tender on palpation and hot to touch
        - inguinal lymphadenopathy
        - urethral discharge
        - features of UTI
        - features of systemic infection
    - chronic bacterial and non-bacterial prostatitis
        - gland feels normal or hard from calcification
- differential
    - cystitis
    - benign prostatic hyperplasia
    - urinary tract stones
    - foreign body in urinary tract
    - bladder cancer
    - prostatic abscess
    - enterovesical fistula
- investigations
    - bloods and cultures if evidence of systemic infection
    - urine culture and microscopy
    - chronic prostatitis
        - NIH CPSI (National Institute of Health Chronic Prostatitis Symptom Index)
            - symptoms suggestive of prostatitis lasting more than 3 months
            - negative urine and prostatic fluid cultures
                - leucocytes present in the prostatic fluid in the inflammatory type, absent in the non-inflammatory type
- management
    - acute prostatitis
        - patient may be acutely unwell and require admission +/- resuscitation
        - analgesia
        - suprapubic catheter may be required for urinary retention
            - urethral catheter may be used if gentle and easy insertion
        - avoid repeat rectal examination (seeding of infection)

        - GUM clinic if likely sexually transmitted
            - screening and contact tracing
        - fluoroquinolones first line
            - e.g. ciprofloxacin, ofloxacin
            - four week course
            - unwell patients may require IV aminoglycosides in addition
    - urology referral:
        - unwell patient or unable to tolerate oral antibiotics
        - inadequate response to oral antibiotics
        - pre-existing urological conditions
            - e.g. obstruction, catheter
        - immunocompromise
        - acute urinary retention
            - requires suprapubic catheter to avoid damaging the prostate
        - investigation for structural abnormalities needed after recovery
    - chronic prostatitis
        - may benefit from analgesia, course of antibiotics and an alpha blocker (e.g. prazosin)
        - require an MDT approach including behavioural therapy/psychology

UC7 UTI/pyelonephritis

- UTI
    - background
        - experienced by half of all females during their lifetime
            - a third have a UTI by age 24
        - UTIs in males suggest structural abnormality
    - categories
        - uncomplicated
            - otherwise healthy young women, not pregnant, with no structural abnormality
        - complicated
            - over 65s
            - men
            - structural or functional abnormality (e.g. obstruction, neurogenic bladder)
            - renal stones
            - catheter
            - pregnancy
            - recent instrumentation
            - co-morbidity (e.g. diabetes, malignancy)
    - pathophysiology
        - most arise from bacteria ascending the urinary tract
        - women are more prone
            - shorter urethra
            - proximity of vagina and rectum to urethra
        - in the majority of patients with uncomplicated cystitis, the causative organism is E coli

- risk factors
  - anatomical abnormality
    - including catheter, stent, instrumentation, neurogenic bladder
    - recurrent acute cystitis
    - advanced age in men
    - nursing home residents
    - neonates
    - comorbidities
      - e.g. diabetes
    - pregnancy
    - immunosuppression
    - advanced neurologic disease
- clinical features
  - dysuria
  - urinary frequency
  - haematuria
  - urgency
  - suprapubic discomfort
- investigations
  - potential can be treated based on history of dysuria and frequency
  - negative urinalysis does not rule out UTI, particularly if pretest probability is high
  - urine dip
    - nitrite sensitivity 81%
      - more specific for diagnosis of UTI
    - leucocyte sensitivity 77%
    - both nitrites and leucocytes 94%
  - urine microscopy
    - usually diagnosed with >10 WBCs per mm^2
  - urine culture
    - gold standard, but not required in all patients
    - culture should be sent for complicated UTIs and pyelonephritis
  - imaging
    - US
      - can reveal complications of UTI such as hydronephrosis and renal abscess
      - probably not required for all cases of pyelonephritis, only when there are complications
    - CT
      - best reserved for renal colic or uncertain diagnosis
- management
  - acute uncomplicated cystitis
    - trimethoprim first line
    - nitrofurantoin second line (must not be used for suspected pyelonephritis as has poor efficacy in upper renal tract)
    - there may be overestimation of resistance in the community as uncomplicated UTIs are rarely cultured

        - acute complicated cystitis
            - usually ciprofloxacin or cephalexin
        - pregnancy
            - asymptomatic bacteriuria is treated in pregnancy
            - UTIs are treated as complex
            - cystitis treatment options include nitrofurantoin, cephalexin, amoxicillin
- pyelonephritis
    - background
        - infection within the renal pelvis
            - usually ascending infection from the bladder
            - can occur by haematogenous spread
        - organisms are usually the same as for UTI
        - repeated attacks can cause scarring and destruction of renal tissue
        - can occur at any age
        - more common in women
    - risk factors
        - structural renal abnormalities
            - including vesicoureteric reflux
        - calculi and urinary tract catheterisation
        - stents or drainage procedures
        - pregnancy
        - diabetes
        - primary biliary cirrhosis
        - immunocompromise
        - neuropathic bladder
        - prostate enlargement
    - presentation
        - usually rapid
            - symptoms appear over a day or two
        - unilateral or bilateral loin pain, suprapubic pain or back pain
        - fever and sometimes rigors
        - malaise, nausea, vomiting, anorexia and sometimes diarrhoea
        - there may be accompanying lower urinary tract symptoms
            - frequency
            - dysuria
            - frank haematuria
            - hesitancy
        - presentation in children can be much less specific
    - investigations
        - urinalysis
            - urine is often cloudy with an offensive smell
            - dipstick may be positive for blood, protein, lecuocytes, nitrites
            - MSU should be sent for microscopy and culture
        - bloods including cultures
        - imaging
            - may be useful in uncertain cases

                - should be done for recurrent cases and probably in men and children
                - US is usually first line
                - contrast CT may be considered for deteriorating cases
    - management
        - supportive
            - rest
            - analgesia
            - fluids
        - admission criteria
            - pregnant women
            - severe vomiting
            - comorbidity such as diabetes
            - signs of sepsis
            - dehydration or inability to take fluids/medication
            - severe pain
            - failure to respond to community management
            - urinary tract obstruction
            - oliguria or anuria
            - suspected complications
            - uncertain diagnosis
            - non-concordance with treatment
            - inadequate access to follow up care
            - relapse as soon as treatment stops
        - antibiotics
            - empirical whilst awaiting culture and sensitivities
            - usually ciprofloxacin or co-amoxiclav for 7 days
            - trimethoprim if cultures confirm sensitivity
        - surgery
            - to drain renal or perinephric abscesses or relieve obstruction
    - complications
        - perinephric abscess
        - sepsis
        - renal abscess
        - acute papillary necrosis
        - AKI or CKD

VASCULAR

VC1 acute limb ischaemia

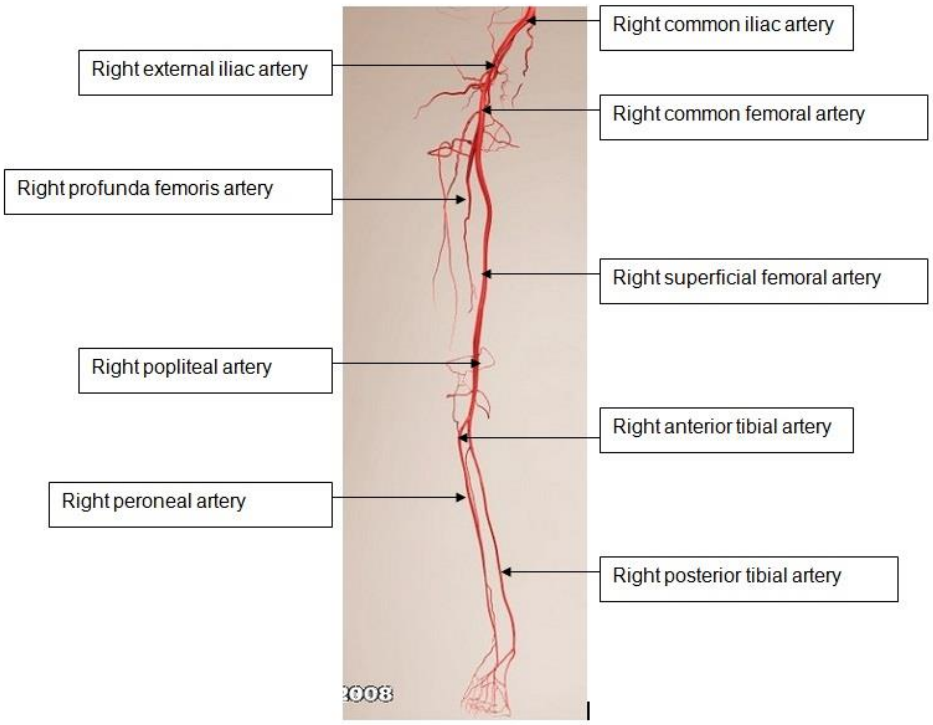
- background
    - any sudden decrease in limb perfusion causing a potential threat to limb viability
        - usually refers to symptoms going on for <2 weeks
        - can include an increase in ischaemic symptoms on a background of claudication
    - incidence around 1 in 6000/year

- incidence may be falling due to recognition and treatment of rheumatic heart disease and atrial fibrillation
- mortality estimates range from 9-22%
- limb salvage is estimated at 70-90%
  - amputations are more common following thrombotic occlusions
- acute upper limb ischaemia is much less common than lower limb
  - usually due to cardiac emboli or from subclavian artery stenosis (listen for subclavian bruit)
  - can be caused by vasculitis or thoracic outlet syndrome
- popliteal aneurysm tend to accumulate thrombus which can dislodge and cause foot ischaemia – patients with even mild ischaemic symptoms and a suspected popliteal aneurysm should be referred to a vascular surgeon urgently
- bilateral limb ischaemia can occur due to saddle embolus at the aortic bifurcation or aortic dissection

- pathophysiology
  - embolus
    - material that travels in the circulation and lodges in a blood vessel somewhere else causing occlusion of the vessel
      - may be solid, liquid or gas
    - upper border is concave (meniscus sign)
    - in acute limb ischaemia, emboli most commonly arise from the heart (80%)
      - usually composed of platelets
      - can also arise from proximal arterial disease (aneurysms or stenoses) and can contain atheroma
        - these are harder to treat as they cannot be thrombolysed
  - thrombosis
    - influenced by Virchow's triad
      - interrupted blood flow
        - venous stasis
        - long surgical operations
        - prolonged immobility
        - varicose veins
      - endothelial or vessel wall injury
        - vessel piercing
        - damage from shear stress or hypertension
        - subsequent contact with procoagulant surfaces
          - bacteria
          - shards of foreign materials
          - biomaterials of implants or medical devices
          - membranes of activated platelets
          - membranes of monocytes in chronic inflammation
      - hypercoagulability
        - hyperviscosity
        - factor V Leiden mutation
        - antithrombin III deficiency
        - protein C or S deficiency
        - nephrotic syndrome

        - changes after severe trauma or burns
        - cancer
        - late pregnancy and delivery
        - race
        - advanced age
        - obesity
        - cigarette smoking
        - hormonal contraceptives
    - rarer causes
        - vasculitis
            - bilateral disease, systemic symptoms, connective tissue disease
        - popliteal entrapment syndrome
            - popliteal artery compressed by gastrocnemius during plantar flexion
            - young men, sporty, pain brought on by exercise
        - compartment syndrome
            - swelling of tissues within fascial compartment compresses artery
                - especially anterior compartment of leg
            - history of trauma
            - pain on passive movement
        - iatrogenic
            - usually injury of common femoral or superficial femoral artery following catheterisation (e.g. coronary angiopathy)
        - aortic dissection
            - dissection flap can occlude the true lumen of a branch vessel causing end organ ischaemia
            - back pain, hypotension, signs changing over time
        - graft occlusion
            - thrombosis of graft, especially if prosthetic rather than vein
                - history of previous vascular surgery
                - severity depends on speed of blockage
                    - may develop collaterals
- clinical presentation (six Ps)
    - pain
        - usually at rest
            - viable limb may present with acute onset short distance claudication
        - rest pain usually worse in most distal part of limb (e.g. toes)
            - may be relieved on dependency (e.g. hanging legs over bed)
        - pain worse on passive movement of the limb is suggestive of compartment syndrome – poor prognostic sign
    - pallor
        - in comparison to opposite limb
        - acutely ischaemic limbs tend to be white rather than blue
        - chronic critically ischaemic limbs may be pink due to compensatory vasodilation (sunset foot)
        - Buerger's test can be useful – pallor on elevation of the limb, erythema on depdendency
    - paraesthesia

        - present in >50%
        - sensory nerves are smaller than motor nerves and more sensitive to ischaemia so tend to be affected first
    - paralysis
        - poor prognostic sign
        - indicates an element of irreversible ischaemia
    - perishingly cold
        - useful when used in comparison to the opposite limb
    - pulselessness
        - checking pulses in unreliable – arterial Doppler should be checked
            - audible signs do not rule out acute ischaemia
- examination



https://www.rcemlearning.co.uk/reference/acute-limb-ischaemia/#1567523577666-20bbdffd-d710

    - full cardiovascular examination
        - arrythmia or valvular heart disease as source of emboli
        - abdomen for aortic aneurysm
    - leg
        - inspection
            - colour
                - white suggests acute ischaemia
                - fixed mottling implies irreversible ischaemia (poor prognostic sign)
                - pink or blue in chronic ischaemia
            - dry gangrene
                - late sign, chronic irreversible ischaemia (>2 weeks)
            - scars

        - from previous surgery
            - midline or transverse AAA repair scar
            - groin scars for EVAR (endovascular abdominal aneurysm repair)
            - vertical scar behind knee from popliteal aneurysm repair
    - palpation
        - compare temperature to opposite leg
            - check other peripheries and core temperature
        - check for femoral pulse
        - tenderness
            - poor prognostic sign
                - suggests ischaemia
        - pain on passive movement
            - urgent vascular review required if compartment syndrome suspected
        - neurological function
            - loss of sensation common
            - loss of motor function poor prognostic sign
    - auscultation
        - Doppler with comparison to other leg
        - ankle brachial pressure index (ABPI) may be useful to help assess severity of ischaemia
            - systolic pressure in pedal arteries divided by brachial artery pressure
                - normal: 1.0-1.2
                - claudication: 0.6-0.8
                - critical ischaemia: 0.2-0.4
            - may be falsely elevated in diabetics due to calcified arteries
- Rutherford's classification
    - I – viable
    - IIa – threatened
        - salvageable if promptly treated
        - intact/slow capillary return
    - IIb – threatened
        - salvageable with immediate reconstruction
        - slow/absent capillary return
        - partial paralysis
    - III – irreversible
        - major tissue loss or permanent nerve damage inevitable
        - capillary return absent + staining
        - profound paralysis
- investigations
    - ECG
        - atrial fibrillation, arrhythmia or acute cardiac event as source of emboli
    - bloods
        - FBC

                - haematological disorder predisposing to thrombosis
            - U&Es
                - dehydration
                - raised potassium secondary to muscle necrosis
            - glucose
                - screen for diabetes
            - CK
                - may be raised if muscle ischaemia has occurred
            - clotting
                - useful to check before prescribing heparin
            - G&S
                - surgery may be required
            - VBG/ABG
                - to look for acidosis secondary to ischaemia
            - imaging
                - choice depends on what is available and vascular advice
                - digital subtraction angiogram can allow therapeutic procedures (thrombolysis, angioplasty)
                - MR and CT angiography are less invasive and provide anatomical information
                - arterial duplex – operator dependent
- management
    - analgesia
        - out of proportion requirement is a red flag for compartment syndrome
        - opiates likely to be required
        - avoid NSAIDs due to increased risk of myocardial events in vasculopaths
    - oxygen
    - heparin
        - 5000 units unfractionated IV immediately for acute limb ischaemia
            - even if surgery or angiography likely
        - prevents propagation of thrombosis
        - IV heparin infusion if definitive treatment is delayed
    - IV fluids
        - manage dehydration
        - in advance of surgery/contrast/reperfusion injury
        - avoid potassium
    - refer
        - urgent referral to vascular specialist
            - heparin infusion during transportation may not be possible
- definitive management
    - Rutherford I
        - admission
        - analgesia
        - oxygen
        - IV heparin infusion
        - formal imaging within normal working hours
    - Rutherford IIa

        - oxygen
        - analgesia
        - heparin
        - ideally immediate imaging
            - in some cases with only minimal sensory loss they may be observed overnight and imaged the next day
    - Rutherford IIb
        - oxygen
        - analgesia
        - heparin
        - imaging before theatre if it does not delay procedure
        - urgent revascularisation
            - operative or thrombolysis
                - embolectomy under local anaesthesia may be attempted if clear history of embolus
                - fasciotomy may be required if limb swells after revascularisation
    - Rutherford III
        - revascularisation likely to kill patient
            - massive released of potassium, CK, myoglobin, lactate, oxygen free radicals
                - can cause renal failure, cardiotoxicity and multi-organ failure
        - options are amputation or palliation
            - patients for whom limb ischaemia is part of the dying process should be palliated rather than subjected to operations
    - thrombolysis
        - alternative to surgery
        - intra-arterial streptokinase or rtPA (tissue plasminogen activator)
            - convert plasminogen to plasmin and lyse thrombin
        - angiogram should be performed afterwards to identify underlying stenosis
        - major complications more common after thrombolysis than surgery (e.g. stroke, major haemorrhage)
        - contraindications:
            - bleeding or severe bleeding tendency
            - pregnancy
            - CVA/TIA <2 months ago
            - intracerebral tumour/AVM/aneurysm
            - surgery <2 weeks ago
            - previous GI bleed
            - trauma <10 days ago

VC2 aortic aneurysmal disease

- definition
    - permanent localised or diffuse dilatation of the abdominal aorta to 1.5 times its normal diameter involving all three layers of the vessel wall

        - normal infrarenal diameters in patients >50 are 1.5cm in women and 1.7cm in men
        - an infrarenal aorta ≥3cm is considered aneurysmal
- types of emergency presentation
    - incidental finding, usually asymptomatic
    - symptomatic or painful aneurysm due to local inflammatory state or acute dissection
    - contained leak (retroperitoneal or compensated rupture)
    - free rupture (uncontained, decompensated rupture with hypovolaemic shock)
    - distal embolization or local thrombosis
- causes
    - strongly associated risk factors
        - male gender
        - age
        - smoking
        - hypertension
        - genetic/familial disposition
            - e.g. connective tissue disorder
    - identified and postulated causes
        - atherosclerotic injury and associated degeneration
        - primary connective tissue degeneration
        - inflammatory arteritis with associated aneurysmal degeneration
        - traumatic injury and pseudoaneurysm
        - mycotic injury and pseudoaneurysm
        - aortic dissection-related aneurysmal degeneration
- pathophysiology
    - true aneurysms involve all three layers
    - pseudoaneurysms are deficient in at least one layer
    - fusiform aneurysms have a bulbous shape and are usually true aneurysms
    - saccular aneurysms have a lateral outpouching and are often false aneurysms
    - 90-95% of AAs are infra-renal
        - extension into the iliac arteries is common
    - pathogenesis
        - infiltration of the vascular wall by lymphocytes and macrophages
        - destruction of elastin and collagen in the media and adventitia by proteases
        - loss of smooth muscle cells with thinning of the media
        - neovascularisation
- clinical assessment
    - palpation of expansile mass
        - two fingers on either side of pulsating mass, observe for separation of fingers with each pulsation
        - may be detectable at 4-5cm depending on body habitus
    - isolated pain in abdomen, epigastrium or back
        - contained leaks tend to present with back pain
    - classic triad of AA rupture
            - pain, typically severe and predominantly in the back
            - signs of circulatory compromise, often frank shock

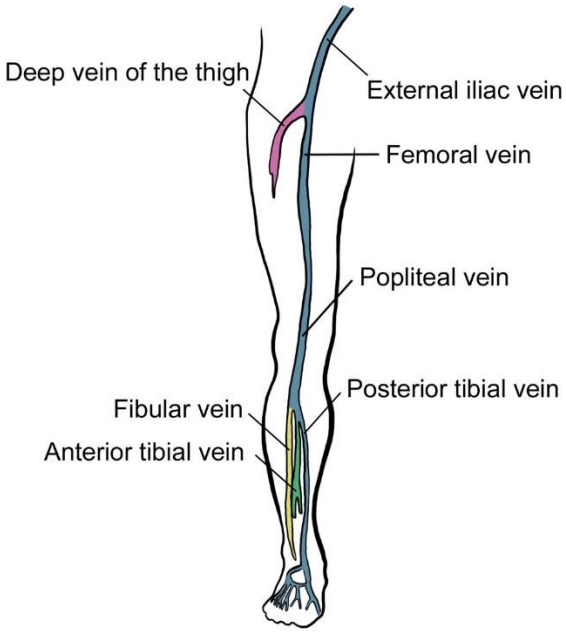
                - presence of pulsatile mass in abdomen
            - triad often not present – e.g. in guarding, obese, hypotensive or large retroperitoneal haemorrhage
        - atypical presentations in the elderly
            - back pain
                - may mimic renal colic
            - radiation of pain to legs
                - mimicking sciatica
            - chronic severe back pain
                - contained rupture
        - tender AAA on palpation is an emergency until proven otherwise
        - massive GI haemorrhage raises suspicion of aorto-enteric fistula
- investigations (depending on presentation)
    - ECG
    - VBG
    - glucose
    - FBC
    - U&E
    - G&S or cross match
    - imaging
        - US
            - can be performed rapidly at the bedside in an unstable patient
            - can detect aneurysm and free fluid
            - AAA diameter measured as maximum diameter in transverse plain
            - around 100% specific and 95% sensitive
        - CT abdomen with contrast
            - best if diagnostic uncertainty
            - shows size and extent of AAA, demonstrates leak and complications
            - relatively stable unstable patients with uncertain diagnosis may still benefit from CT with ongoing resuscitation
        - MRI
            - highly specific and sensitive
            - only for elective settings
        - x-ray
            - may demonstrate mural calcification
- management
    - rupture
        - 2x large peripheral IV cannulae
            - giving sets with hand pumps or rapid infusers
        - target systolic of 90mmHg to maintain end organ perfusion
        - cross match 6 units, consider activating massive haemorrhage protocol
        - FBC, U&E, coag, VBG, ECG
        - titrated analgesia
        - urgent transfer to operating theatre unless palliation appropriate
            - involve vascular surgery, anaesthetics, ITU
        - urinary catheter and arterial line unless this will delay transfer
        - contained rupture still requires urgent repair

- acute pain
    - refer to vascular surgeon urgently
        - even in stable patient without rupture
    - assume that pain in a patient with a AAA is due to an aortic emergency until proven otherwise
- incidental finding
    - refer to vascular surgeon if potential surgical candidate – urgency depends on size and presence/absence of symptoms
        - <2cm normal
        - 2-5.5cm and asymptomatic requires regular US follow up
        - >5.5cm (5.0cm in female) requires operative intervention in patients suitable for surgery
- repair options
    - open repair
        - only option for ruptured AAA
        - abdominal or flank incision, vessels above and below aneurysm are controlled, aneurysm sac is opened with placement of synthetic graft
        - similar mortality to endovascular repair in the long term
        - around 20% require a subsequent operation
    - endovascular stent grafting
        - less invasive with lower perioperative morbidity and mortality
        - involves intraluminal introduction of covered stent through the femoral and iliac arteries
        - can be performed under local anaesthesia
        - only possible in around 50% of unruptured aneurysms
        - small risk of aneurysmal sac reperfusion and late rupture
        - 20-30% require secondary intervention in following 6 years

- prognosis
    - asymptomatic
        - risk of rupture increases with size, but any size can rupture
        - >6cm has 5 year survival rate of around 20% and 1 year rupture risk of around 50%
        - mortality rate is decreased by elective repair
    - ruptured AAA
        - 100% fatality unless repaired
        - of those surviving to hospital, mortality for surgical repair is still high (around 50%)
        - mortality correlates with age, comorbidities and hypotension

VC3 DVT

- background
    - incidence of 100-200 per 100,000
        - 2.5.-5% of people affected at some point in their lifetime
    - up to 50% have long term consequences
        - early diagnosis and management reduces adverse outcomes
- anatomy and physiology



https://www.rcemlearning.co.uk/reference/deep-vein-thrombosis/#1568729656062-fc7bc64c-6a60

- anatomy
  - venous anatomy of the leg has low flow areas such as soleal sinuses, valve pockets and venous confluences that predispose to thrombosis
  - lower limbs are also more prone to dependent effects of gravity
  - detectable clot is usually found in the distal venous circulation:
    - anterior tibial vein
    - posterior tibial vein
    - peroneal vein
  - clot can then propagate proximally into the popliteal, femoral and iliac veins
- pathophysiology
  - Virchow's triad (generally more than one factor required)
    - venous stasis
    - hypercoagulable state
    - endothelial vessel wall damage
  - venous stasis
    - atrial fibrillation
    - left ventricular dysfunction
    - immobility or paralysis
    - venous insufficiency or varicose veins
    - venous obstruction from tumour, obesity or pregnancy
  - hypercoagulable state
    - malignancy
    - pregnancy and peri-partum period
    - oestrogen therapy
    - trauma or surgery of lower extremity, abdomen or pelvis
    - inflammatory bowel disease
    - nephrotic syndrome
    - sepsis

                - thrombophilia
            - vascular wall injury
                - trauma or surgery
                - venepuncture
                - chemical irritation
                - heart valve disease or replacement
                - inflammatory bowel disease
                - atherosclerosis
                - indwelling catheters
            - a process is triggered resulting in local cytokine production and leucocyte adhesion to the endothelium
- upper limb DVT
    - risk factors:
        - central venous catheters
        - malignancy
        - inherited/acquired thrombophilia
        - pacemakers
        - upper limb surgery and/or immobilisation
- clinical presentation
    - pain
        - in approximately 50%
    - swelling/oedema
        - usually unilateral
        - ischaemic change is rare
    - warmth and erythema
        - can occur over the area of the thrombus
    - local tenderness
        - frequently present
    - respiratory symptoms
        - first presentation may be that of a PE
- differential
    - ruptured popliteal synovial membrane or cyst (Baker's cyst)
    - ruptured calf muscles or tendons
    - severe muscle cramp
    - cellulitis
    - lymphoedema
    - arterial insufficiency
- investigations
    - d-dimer
        - detects fibrin fragments from clot degradation
        - high sensitivity when used in appropriate population
            - negative d-dimer results in 3 months incidence of subsequent DVT of around 0.5%
        - specificity is poor, raised in any condition with clot turnover, e.g.:
            - infection
            - trauma
            - haemorrhage

                - cancer
                - post-surgery
            - becomes increasingly positive with advancing age
                - NICE recommend age adjusted threshold
                    - 10x age for patients over 50 (or 5x age depending on assay used – should be confirmed with laboratory prior to using the rule)
            - levels remain detectable for 7 days after initial clot formation and false negatives may occur outside this time period
            - always used to rule out, not rule in
            - pregnant and post-partum women and IVDUs should always be considered high risk for DVT so d-dimer is unhelpful for these groups
- risk stratification
    - NICE recommends the Two-level DVT Wells Score
        - active cancer (treatment ongoing, within 6 months or palliative) (1)
        - paralysis, paresis or recent plaster immobilisation of lower extremities (1)
        - recently bedridden for more than 3 days or major surgery within 12 weeks requiring general or regional anaesthesia (1)
        - localised tenderness along the distribution of the deep venous system (1)
        - entire leg swollen (1)
        - calf swelling 3cm larger than asymptomatic side (1)
        - pitting oedema confined to the symptomatic leg (1)
        - collateral superficial veins (non-varicose) (1)
        - previously documented DVT (1)
        - alternative diagnosis is at least as likely as DVT (-2)
        - DVT 'likely' if 2 or more points
            - pre-test probability of around >30% before d-dimer
            - no indication for d-dimer because post-test probability of negative d-dimer still too high to exclude DVT
            - require imaging
            - if Duplex US negative, d-dimer should be checked
                - if positive, anticoagulation should be stopped and US repeated in 6-8 days
                - if negative, consider other diagnoses
        - DVT 'unlikely' if 1 point or less
            - low/moderate risk
            - pre-test probability of <15%
            - can exclude DVT and discharge patient if d-dimer negative
            - if d-dimer positive, will need imaging
- imaging
    - Duplex ultrasonography
        - occlusion of vascular lumen is major criterion for assessing clot presence
        - loss of normal phasic signal from venous blood flow also suggests presence of clot
        - clot proximal to inguinal ligament cannot be visualised on US

        - NICE guidelines are that interim dose of anticoagulant should be given if US not available within 4 hours of being requested
        - indeterminate US may require further imaging such as venography if it is important to make a firm diagnosis
    - impedance plethysmography
        - measures small changes in electrical resistance of the calf
            - reflects blood volume changes, indirectly indicating presence of thrombus
        - insensitive for non-occluding thrombus, calf thrombus or thrombus above the inguinal ligament
    - MRI
        - sensitivity and specificity approaches that of venography
        - highly sensitive for calf thrombus and thrombus above the inguinal ligament when CT not practical
        - useful in pregnancy where Doppler flow characteristics change with the gravid uterus
        - limited availability of scanners
    - CT
        - effective in diagnosis of iliofemoral and more proximal thrombus
        - problems with contrast reactions, radiation exposure and presence of metal implants
    - venography
        - previously the gold standard, replaced with non-invasive and less harmful techniques
- management
    - bridging LMWH and fondaparinux
        - 4-5 day bridging regime whilst loading with warfarin
        - until INR >2 for 24 hours
    - warfarin
        - inhibits vitamin K dependent synthesis of biologically active forms of calcium dependent clotting factors (II, VII, IX, X)
        - tendency towards paradoxical prothrombotic state in first few days, hence requirement for bridging therapy
        - warfarin should be continued for 3 months (unprovoked) and 6 months or longer in cancer patients or other ongoing risk factors
    - thrombolysis
        - rarely used in UK
        - local catheter directed thrombolysis recommended by NICE in specific circumstances:
            - iliofemoral DVT with symptoms for less than 14 days, good functional status, life expectancy of >1 year, low risk of bleeding
        - NOACs
            - rivaroxaban and apixaban are direct Xa inhibitors
            - rivaroxaban
                - 15mg BD for 21 days then 20mg OD
            - apixaban
                - 10mg BD for first 7 days then 5mg BD

        - mechanical treatments
            - compression stockings
                - effective in reducing effects of post-thrombotic syndrome
                - below knee graduated stockings for at least 2 years, starting one week after diagnosis
        - vena caval filters
            - can reduce risk of recurrent PE to 4%
            - management option for patients with contraindications to anticoagulation or recurrent DVTs on warfarin
- VTE prophylaxis in ED
    - recommended for lower leg casts if significant transient risk of VTE and any permanent risk factors for VTE and no contraindications
    - significant transient risk of VTE (any)
        - rigid immobilisation in plaster cast
        - non weight-bearing status
        - acute severe injury (dislocation, fracture or complete tendon rupture)
    - permanent risk of VTE (any)
        - current hormone therapy (COCP, HRT, tamoxifen)
        - personal or first degree relative VTE history
        - active smoker
        - any recent hospital admission/major surgery
        - pregnant or immediately post-partum
        - any serious medical co-morbidity including cardiac failure, COPD, chronic renal failure, inflammatory bowel disease
        - extensive varicosities
        - active cancer
        - obesity (BMI >30)
        - known thrombophilia
        - age >60
    - relative contraindication (any) – discuss with haematologist
        - haemophilia/other haemorrhagic disorder
        - thrombocytopaenia or previous HIT
        - recent cerebral haemorrhage or severe hypertension
        - active peptic ulcer/recent GI bleeding
        - recent major trauma/surgery to eye or nervous system
        - hypersensitivity to any form of heparin
        - known eGFR <30ml/min
        - risk deemed to outweigh benefits by clinician
    - if thromboprophylaxis advised:
        - baseline eGFR and/or platelet count for patients with suspected or known renal impairment and/or thrombocytopaenia
        - any patient with baseline moderate or worse renal impairment (eGFR <50ml/min) to be dose adjusted as per BNF/pharmacist advice
        - prophylactic LMWH once daily until date of clinical/orthopaedic review
        - patient educated in subcutaneous injection technique or district nurse referral for ongoing injection
        - safety net regarding bleeding complications

        - written guidance for patient and GP regarding signs of HIT/coagulopathy and advice to consider platelet check in 5 days if review delayed

OTHER CLINICAL PRESENTATIONS

XC1 major incident management

- background
    - declared when local services have the potential to be overwhelmed
- planning for disaster
    - The Civil Contingencies Act 2004
        - provides a framework for civil protection in the UK
        - sets out roles and responsibilities for those involved at local level
            - category 1 responders
                - includes emergency services, local authorities, NHS bodies
                - responsible for planning for emergency situations and putting the plans into action
                - responsible for liaising with the public and other responders and for business continuity management
            - category 2 responders
                - includes the Health and Safety Executive, transport and utility companies
                - only tend to be affected when the incident affects their sector but will liaise with category 1 responders to provide assistance
            - category 1 and 2 responders come together to form Local Resilience Forums, based on police areas
        - allows the making of special temporary legislation to help deal with the most serious of emergencies
            - use of emergency powers is a last resort option and it should not be assumed that emergency powers will be available
    - disaster plans should include
        - generic plans
        - specific plans (relating to specific emergencies)
        - plans where there is single vs multilevel agency involvement
        - multilevel planning (covering more than one area of government)
    - testing the plan
        - plans should be regularly tested and updated, involving all those involved at a strategic level as well as senior frontline staff
            - training exercises may be discussion based, tabletop or 'live'
            - they help the team gain familiarity with equipment and each other
- reporting of a major incident
- major incident plan
    - a typical plan has several parts
        - core major incident plan
            - sets out the organisational arrangements for major incidents and explains them in detail

- having a written major incident plan is a legal requirement and part of the NHS guidance on emergency planning
- is the official organisational policy on major incident planning and a reference document for staff on the actions required
- if a major incident has been declared, if you have not read the plan, this is not the time to start
- notification of incidents
    - usually involves switchboard being informed by the ambulance service
        - the telephonist takes the details and verifies by calling the ambulance control room back
    - once confirmed, key members of staff are notified through a call-out cascade
- messages received by switchboard may be:
    - major incident standby
        - a core group of management and operational staff are informed
        - allows them to prepare for activation
    - major incident declared, activate plan
        - the ambulance service requires the hospital to move to its full major incident response
        - the Emergency Department is cleared and staff and equipment made ready to deal with patients
    - major incident cancelled
        - sent if it becomes clear that a response is not required after assessment on scene
    - major incident stand down
        - issued within the hospital once the hospital response is complete
        - will come from the hospital control team

- action cards
    - explain what each person's role involves when a major incident is declared
    - staff are allocated to roles
    - the role is more important than the person doing it
    - there are a list of specific tasks that a person performing a specific role must do
    - there is a card for each key role identified in the major incident plan
    - most include space for logging the time when actions are completed
    - when a more senior or appropriate person arrives, the role and action card may be handed over and the original person redeployed elsewhere
- specialist plans
    - deal with specific issues
    - e.g. incidents involving hazardous chemicals, children, large amounts of patients with burns

- METHANE

        - **m**ajor incident declared
        - **e**xact location
        - **t**ype of incident
        - **h**azards
            - present and potential
        - **a**ccess
            - routes that are safe to use
        - **n**umber, type and severity of casualties
        - **e**mergency services now present and those required
- organisation of response
    - strategic
        - Chief Executive and Trust board level
        - the strategic team has the ability to approve release of funds for equipment and supplies
        - they work with other regional agencies to oversee the impact of the incident on the wider community
        - also known as gold command
    - tactical
        - oversee and coordinate teams providing patient care
        - maintain an overview of the situation within the hospital
        - can make decisions about the allocation of resources
        - liaise with tactical teams in other services to share information
        - also known as silver level
    - operational
        - front line teams involved directly in patient care or providing essential resources to allow this to happen
        - also known as bronze level
        - types of team include:
            - ED triage team
                - usually senior nurse and doctor at ED entrance
            - treatment teams
                - provide direct clinical care
                - usually one or more teams for each category of immediate, delayed and minor
                - often involve a senior doctor and nurse overseeing a number of other doctors and nurses
            - theatre teams and medical teams
                - deliver definitive care
            - other support service teams
                - e.g. labs, radiology, catering, portering, blood transfusion
- mass casualty triage
    - background
        - a medical mass casualty incident is any situation in which medical resources such as personnel or equipment are overwhelmed by the number and severity of casualties
        - effective triage allows ordered prioritisation in a chaotic situation
        - triage sieve

        - initial primary assessment
        - takes place on scene
        - should take no more than 30 seconds per patient
        - aims to rapidly identify patients who need a life saving intervention
        - designed to be rapid, reliable and reproducible irrespective of the provider performing it
    - triage sort
        - secondary triage process
        - typically takes place in a more permissive environment such as a casualty clearing area
        - performed by a more experienced clinician
- triage categories
    - red (P1)
        - immediate
        - casualties in need of life-saving interventions and requiring immediate treatment
    - yellow (P2)
        - urgent
        - unwell but with a degree of stability
        - unable to walk but have comparatively normal physiology
        - typically require treatment/interventions within 24 hours
    - green (P3)
        - delayed
        - 'walking wounded'
        - able to ambulate to a triage area
        - deemed safe to have a treatment delay of 4 hours or more
    - blue (P4)
        - expectant
        - casualties with such severe injuries that they are not expected to survive with the resources available and their treatment would divert effort away from those with a greater chance of survival
        - invoking the P4 category requires Gold authority (usually ministerial level) in a UK incident
        - it is an ethically and emotionally difficult category and only used where resources remain overwhelmed despite implementation of the normal mass casualty plan
        - if adequate resources become available they are treated as P1
        - World Medical Association advice:
            - 'it is unethical for a physician to persist, at all costs, at maintaining the life of a patient beyond hope, thereby wasting to no avail scarce resources needed elsewhere'
    - black (or white)
        - dead
- triage cards
    - should be attached to the patient showing their triage category
    - some have space for basic note keeping

        - when triage cards are not available patients can be labelled with their category number on their forehead
    - reassessment
        - triage is a dynamic process and regular re-assessment is required
    - primary triage
        - aims to identify patients in need of life-saving procedures by other healthcare providers
        - there are two exceptions to the 'triage not treatment rule'
            - provision of basic airway manoeuvres/adjuncts if the patient is not breathing
            - application of a Combat Application Tourniquet (CAT) for catastrophic haemorrhage
        - MPTT-24
            - Modified Physiology Triage Tool
            - can be effectively delegated to any competent person at the scene and does not require high level medical training
            - team members with less medical knowledge may be more likely to comply with the protocol and less likely to be distracted by other tasks or interventions
            - may miss patients at risk of rapid deterioration but wholly salvageable (e.g. a walking patient with airway burns)
            - tool questions
                - catastrophic haemorrhage?
                    - yes
                        - apply tourniquet or haemostatic dressing
                        - P1
                - walking?
                    - yes
                        - P3
                - breathing?
                    - no
                        - dead
                - responds to voice
                    - no
                        - put in recovery position
                        - P1
                - respiratory rate 12-23
                    - no
                        - P1
                - heart rate <100
                    - no
                        - P1
                    - yes
                        - P2
    - secondary triage
        - a secondary, more detailed triage is performed when time and resources allow

        - may be at the scene but usually at the casualty clearing station or the hospital
        - clinician judgement is allowed
        - triage sort
            - most common method used
            - taught by MIMMS
            - three physiological variables are measured and scored and the some of the scores gives the triage category
                - X – Glasgow coma scale
                    - 13-15 = 4
                    - 9-12 = 3
                    - 6-8 = 2
                    - 4-5 = 1
                    - 3 = 0
                - Y – respiratory rate
                    - 10-29 = 4
                    - >29 = 3
                    - 6-9 = 2
                    - 1-5 = 1
                    - 0 = 0
                - Z – systolic blood pressure
                    - ≥90 = 4
                    - 76-89 = 3
                    - 50-75 = 2
                    - 1-49 = 1
                    - 0 = 0
            - 12 = P3
            - 11 = P2
            - ≤10 = P1
            - 0 = dead
            - priority should be upgraded depending on the injury/diagnosis
    - paediatric triage
        - children are often over-triaged at the expense of adults, either because an adult triage tool is used or because of emotional involvement
        - paediatric triage tape
            - measures from heel to top of head and is read off at the appropriate level
            - estimated weight and adjusted triage sieve algorithm values are provided
        - JumpSTART algorithm
            - advocated in the 2018 NHS England Clinical Guidelines for Major Incidents
            - may be used for any patient who appears to be a child
            - algorithm questions
                - able to walk?
                    - yes
                        - minor

                - secondary triage when possible
        - not able to walk; spontaneous breathing?
            - no
                - position airway
                    - if spontaneous breathing – immediate
                    - if no spontaneous breathing and no pulse – expectant
                    - if no spontaneous breathing and a palpable pulse, give 5 rescue breaths
                        - apnoea – expectant
                        - spontaneous breathing – immediate
        - spontaneous breathing; respiratory rate 15-45?
            - no
                - immediate
        - respiratory rate 15-45; palpable pulse?
            - no
                - immediate
        - palpable pulse; neurological assessment (AVPU)
            - inappropriate P (e.g. posturing) or U
                - immediate
            - A, V or appropriate P (e.g. withdrawal from painful stimulus)
                - delayed
- chemical, biological, radiological and nuclear incidents
    - may not present immediately; can be an emerging picture over time
        - could be hours to weeks
    - cases may be noted for:
        - similar epidemiological features
        - geographical grouping
        - severity of symptoms or signs
        - unusual aetiology given circumstances (e.g. a disease not normally seen in the UK in a patient with no travel history or contacts)
        - poor response to conventional treatment
        - unknown aetiology

XC2 PHEM

- FPHC skills framework
    - level A – first aider certified by a non-national organisation
    - level B - first level responder, nationally certified and qualified to meet statutory requirements within the workplace
    - level C – nationally certified pre-hospital responder (use of airway adjuncts and oxygen) e.g. Community First Responder

    - level D – nationally certificated non-healthcare professional pre-hospital provider caring for patients as a secondary role (e.g. police officers in specialist roles, fire service)
    - level E – nationally certificated non-healthcare professional prehospital provider caring for patients as a primary role (e.g. UKSAR)
    - level F – non-registered healthcare professional (e.g. Ambulance Technician) working at level 6/7 Skills for Health
    - level G – registered pre-hospital care practitioner working at level 7/8
    - level H – advanced registered prehospital care practitioner working at level 8
- crew resource management
    - training procedures for use in environments where human error can have devastating effects
    - focuses on interpersonal communication, leadership and decision-making
    - five-step assertive statement process
        - opening or attention getter
            - using the person's name or title
        - state your concern
        - state the problem as you see it
        - state a solution
        - obtain agreement
            - 'does that sound good to you?'
- trapped patients
    - the Fire Service provides expertise and personnel
    - plan A
        - done with care and staged
        - due attention to spinal care
    - plan B
        - threat to life is severe and patient is pulled out quickly (e.g. vehicle fire, cardiac arrest)
        - there is less attention to spinal care
- ATMISTER handover
    - **a**ge of patient
    - **t**ime of injury
    - **m**echanism of injury
    - **i**njuries present and suspected
        - follow <C> ABCDE format
    - **s**igns including physiological parameters
    - **t**reatment given and needed
        - use <C> ABCDE format for treatment given
    - **e**stimated time of arrival
    - **r**equests (e.g. blood, RSI, theatre)
- musculoskeletal injuries requiring direct transfer to major trauma centre
    - major long bone injuries (femur, tibia, humerus) with:
        - bone protruding out of skin
        - loss of skin greater than the size of a credit card

        - absence of pulses or compromise in capillary refill distal to a suspected fracture not rapidly recovering once the limb reduced into anatomical alignment
        - severe soft tissue damage to limbs with or without fractures
- Recognition Of Life Extinct (ROLE)
    - allows paramedics to recognise death and not start resuscitation
    - situations are:
        - decapitation
        - massive cranial and cerebral destruction
            - where injuries considered to be incompatible with life
        - hemicorporectomy (or similar massive injury)
        - decomposition/putrefaction
        - incineration
            - presence of full thickness burns with charring of >95% of the body surface
        - hypostasis
            - pooling of the blood in congested vessels in the dependent part of the body in the position in which it lies after death
        - rigor mortis
        - foetal maceration in a newborn child
            - a neonate born with such severe abnormalities it is considered incompatible with life
- termination of resuscitation
    - where there is a doctor, paramedic or registered nurse in attendance
    - full advanced life support has been provided and the patient does not respond and remains asystolic after 20 minutes
    - also if all of the following are met:
        - >15 minutes since the onset of collapse
        - no bystander CPR prior to the arrival of the ambulance
        - absence of any exclusion factors
        - asystole for >30 seconds on the screen
        - no pulse (assessed over 30 seconds)
        - no respiratory effort (assessed over 30 seconds)
        - fixed dilated pupils
        - absent heart sounds
- activation of prehospital services
    - immediate dispatch criteria (London HEMS)
        - fall from >2 floors (>20 feet)
        - ‘one under’ (fell or jumped in front of a train)
        - ejected from vehicle
        - death of a same vehicle occupant
        - amputation above wrist or ankle
        - trapped under vehicle (not motorcycle)
        - request from any other emergency service
    - incident categories for interrogation (London HEMS)
        - shooting
        - stabbing

        - explosions
        - road traffic collisions
        - industrial accidents/incidents
        - hanging
        - drowning
        - entrapments
        - amputations
        - burns/scalds
        - building site accidents
        - falls from height <2 floors
        - impalement
- life-threatening medical conditions requiring intervention in the primary survey
    - A
        - actual or impending airway obstruction
            - airway manoeuvres/adjuncts
            - suction
            - RSI
            - surgical airway
            - adrenaline if anaphylaxis
    - B
        - tension pneumothorax
            - decompression +/- thoracostomy
        - heart failure
            - GTN, CPAP if available
        - asthma/COPD
            - nebulisers (salbutamol, ipratropium, magnesium)
            - titrated oxygen therapy
        - poor tidal volumes
            - NIPPV
            - RSI and IPPV
        - hypoxaemia
            - titrated oxygen therapy
        - cardiac tamponade
            - pericardiocentesis +/- thoracotomy
    - C
        - haemodynamic instability
            - IV fluids
            - inotropes
            - vasopressors
        - cardiac arrest
            - chest compressions
    - D
        - low blood sugar
            - sugar
        - decrease GCS
            - airway management/IPPV if indicated
    - E

        - sepsis
            - antibiotics
- airway
    - prediction of difficult airway (HAVNOT)
        - **h**istory of previous airway difficulties
        - **a**natomical abnormalities of the face, mouth, teeth
        - **v**isual clues (e.g. obesity, facial hair, age >55)
        - **n**eck immobility
        - **o**pening of mouth <3 fingers
        - **t**rauma (e.g. maxillofacial injury, burns, airway bleeding)
    - prediction of difficult airway rescue
        - difficult mask ventilation (MOANS)
            - **m**ask seal difficulty (beard, facial trauma)
            - **o**besity/**o**bstetric/**o**bstructed
            - **a**dvanced age
            - **n**o teeth
            - **s**norer/**s**tiff lungs (e.g. severe asthma, pulmonary oedema)
        - difficult supraglottic airway (RODS)
            - **r**estricted mouth opening (<4-5cm/3 fingers)
            - **o**bstruction at the larynx or below
            - **d**istorted airway (affects seal)
            - **s**tiff lungs/stiff c-spine
        - difficult cricothyroidotomy (SHORT)
            - **s**urgery/**s**cars/**s**hort neck
            - **h**aematoma
            - **o**besity
            - **r**adiotherapy
            - **t**rauma (laryngeal)/**t**umours
- overview of pre-hospital anaesthesia
    - RSI indicated
        - airway compromise
        - respiratory failure
        - unconsciousness
        - unmanageable/agitated head injured patients
        - humanitarian indications
    - preparation
        - kit dump
            - monitoring
            - oxygen
            - suction
            - laryngoscope with different blades
            - spare laryngoscope
            - bougie
            - ETT (+smaller) – cuffs tested
            - circuit
                - catheter mount
                - HME filter

        - ETCO2 detector
      - syringe
      - tube tie/adhesive tape
      - BVM +/- mechanical ventilator
      - difficult airway equipment
        - supraglottic airway
        - surgical airway kit
      - drugs + spare
    - patient positioned with 360° access
    - monitoring attached
    - venous access
    - team briefed
  - pre-induction
    - pre-oxygenation with airway adjuncts
    - run through of equipment checklist
    - collar and head blocks removed
  - induction
    - RSI drugs administered
    - cricoid pressure applied
  - intubation
  - post intubation
    - breathing circuit connected
    - ETT placement confirmed and secured
    - collar and head blocks replaced
    - all procedures completed
    - patient packaged
    - ventilation adjusted for normocapnia
    - anaesthesia maintained
    - sufficient oxygen
  - transfer
    - maintenance of anaesthesia
    - continual assessment and monitoring
    - supporting equipment
    - contemporaneous notes
  - problems
    - can't see cords
      - optimisation measures
        - cricoid manipulation
        - release cricoid pressure
        - insert blade to maximum and slowly withdraw
        - suction
        - adjust patient and/or operator position
        - change blade (e.g. long or McCoy)
        - change operator
    - failed intubation
      - options:
        - insert supraglottic device

        - perform surgical airway
        - BVM to hospital
        - wake and allow to spontaneously ventilate +/- sedation
- consideration of reversible causes in pre-hospital cardiac arrest
    - hypoxia
        - is the oxygen cylinder full?/is the wall mount properly connected?
        - is the oxygen turned on?/is there a leak?
        - is the oxygen tubing attached?/is it kinked?
        - does the bag valve resuscitator have a reservoir?/is it inflated?
        - is the bad valve resuscitator intact?/ is the pressure release closed?
        - is the face mask size appropriate?/is the advanced airway correctly placed
        - is the seal adequate?/is the cuff adequately inflated?
        - is the mouth clear of secretions, blood or vomitus?/is there a foreign body?
        - is the patient cyanosed?/is a CO2 detector available?
        - is the chest visibly rising?/is it symmetrical?
        - is the air entry adequate?/is it equal bilaterally?
    - hypovolaemia
        - history of vomiting and/or diarrhoea?
        - visible bleeding?/occult bleeding in the chest, abdomen, pelvis, limbs?
        - evidence of distributive shock (anaphylactic, septic, neurogenic)?
    - hypothermia
        - is a low reading thermometer being used?
        - was a core temperature reading taken?
    - hypo/hyperkalaemia
        - ECG – small T-waves or large U-waves suggestive of low potassium?
        - ECG – large peaked T-waves and absent P-waves (suggesting high potassium)?
    - H+ imbalance
        - high pCO2 – is intubation or chest tension decompression indicated?
        - low pCO2 – with pH <7.0, sodium bicarbonate may be indicated
    - tension pneumothorax
        - has needle decompression been performed?
        - has an intercostal drain been inserted and is it draining adequately?
    - tamponade
        - are the ECG QRS complexes small or alternating in amplitude?
        - is sonography available to confirm and to facilitate decompression?
    - toxins
        - antidotes or prolonged resuscitation may be indicated
        - is the patient taking any drugs (orally, rectally, IM or IV)?
        - has the patient been given any medications recently?
        - are any medications present in the patient's pockets?
        - is there evidence of needlestick or puncture wounds?
        - is there evidence of chemical contamination?
    - thrombosis (cardiac)
        - ischaemic changes on ECG?
        - risk factors for coronary artery disease?
    - thrombosis (pulmonary)
        - swollen calf?

        - history of inactivity or peripheral vascular disease?
- pre-hospital communication
    - basic model for communication
        - the sender and receiver
        - the message itself
        - the medium of the message
        - contextual factors such as timing and environmental factors
        - feedback
    - emergency operations centre
        - each regional ambulance service has an emergency operations centre where 999 or 112 calls requesting an ambulance are triaged
        - the Advanced Medical Priority Dispatch System (AMPDS) is used to assign a code, which has a priority allocated to it with a timeframe for response
        - clinical advice given by call handlers is generated by the automated system and they are not clinicians
        - each centre also has a trauma desk to allocated resources such as HEMS, advanced paramedics, BASICS clinicians or MERIT teams
    - Airwave
        - communications system used in England, Scotland and Wales (Barracuda in Northern Ireland) based on the terrestrial trunked radio (TETRA) specification
        - it is a hybrid of VHF and mobile phone technology and can withstand high numbers of users
        - it is highly secure with encryption to prevent eavesdropping
        - the units have emergency buttons which will override other activity
    - other communication options
        - radio
        - mobile phone
        - hand signals
        - runners
    - radio etiquette
        - listen before transmitting to ensure you do not cut across another user
        - pause for one second after pressing the push-to-talk button – there is often a delay and the first part of the message may be lost otherwise
        - speak across the microphone rather than into it to reduce additional sounds
        - speak at normal volume – shouting can lead to distortion
        - maintain a normal rhythm and speak clearly and distinctly; send the message phrase by phrase rather than word by word
        - use a slightly higher pitch as it transmits better, but avoid extremes and variations of pitch
        - messages should be brief and to the point with no unnecessary talk
    - NATO alphabet
        - A – Alpha
        - B – Bravo
        - C – Charlie
        - D – Delta
        - E -Echo
        - F – Foxtrot

        - G – Golf
        - H – Hotel
        - I – India
        - J – Juliet
        - K – Kilo
        - L – Lima
        - M – Mike
        - N – November
        - – Oscar
        - P – Papa
        - Q – Quebec
        - R- Romeo
        - S – Sierra
        - T – Tango
        - U – Uniform
        - V – Victor
        - W- Whiskey
        - X- X-ray
        - Y – Yankee
        - Z – Zulu
    - numbers
        - 0 – zero
        - 1 – wun
        - 2 – too
        - 3 – tree
        - 4 – fower
        - 5 – fife
        - 6 – six
        - 7 – seven
        - 8 – ait
        - 9 – niner
    - common words and phrases
        - radio check
            - to ask about signal strength and readability of the transmission
            - response could be a number from 1 (bad) -5 (excellent) or ‘loud and clear’, ‘weak but readable’, ‘weak and distorted’, ‘strong but distorted’
        - yes and no
            - sometimes doubled for clarity (‘yes yes’, ‘no no’)
        - over
            - to end transmission in an ongoing conversation
        - out
            - to end a conversation
            - only needs to be said by one person, usually by control
        - roger
            - ‘I received your last transmission satisfactorily’
        - say again

                - ‘I did not receive or understand your last message, please say again’
            - figures
                - ‘I am about to read a series of numbers’
            - I spell
                - ‘I am about to spell a word’
            - read back
                - ‘please read back to me what I just told you’
            - wait
                - ‘I have received your message, give me a second to reply’
            - standby
                - ‘I am too busy to take your call right now but will call you back later’
            - priority
                - ‘I have a priority message and need to interrupt someone else to send it’
                - wait for a gap then transmit ‘priority, priority’ followed by your call sign and wait for control to reply before passing the message

XC3 safeguarding in adults

- background
    - adult abuse is defined as a single or repeated act or lack of appropriate actions, occurring within any relationship where there is an expectation of trust, which causes harm or distress to a vulnerable person
    - safeguarding adults is defined by the Care Act (2014) as:
        - ‘protecting an adult’s rights to live in safety, free from abuse and neglect’
    - the department of health defines a vulnerable adult as a person aged 18 or over who is or may be in need of community care services by reason of mental or other disability, age or illness and who is or may be unable to take care of him or her self, or unable to protect him or her self against significant harm or exploitation
- risk factors for abuse
    - lack of mental capacity
    - increasing age
    - being physically dependent on others
    - low self-esteem
    - previous history of abuse
    - negative experiences of disclosing abuse
    - social isolation
    - lack of access to health and social services or high quality information
- types of abuse (Care Act 2014)
    - physical abuse
        - may involve physical violence, misuse of medication, inappropriate restraint or sanctions
    - sexual abuse
    - psychological abuse
        - including emotional abuse, threats of harm or abandonment, deprivation of contact, humiliation, blaming, controlling, intimidation, harassment, verbal abuse
    - financial or material abuse

        - including theft, fraud, exploitation, pressure in connection with wills, property, inheritance or financial transactions, misuse or appropriation of property, possessions or benefits
    - neglect and acts of omission
        - including ignoring medical or physical care needs, failure to provide access to appropriate health, social care or educational services, withholding medication, adequate nutrition and heating
    - discriminatory abuse
        - including racist, sexist abuse or abuse based on disability
    - domestic abuse
        - including psychological, physical, sexual, financial, emotional abuse, so-called 'honour-based' violence
    - modern slavery
        - includes slavery, human trafficking and forced labour or domestic servitude
    - organisational abuse
        - including neglect and poor care practice within an institution or specific care setting such as a hospital or care home
    - self-neglect
        - includes a wide range of behaviour neglecting to care for personal hygiene, health or surroundings and includes behaviour such as hoarding
- Department of Health six principles of good safeguarding practice
    - empowerment
        - presumption of person-led decisions and informed consent
    - protection
        - support and representation for those in greatest need
    - prevention
        - it is better to take action before harm occurs
    - proportionality
        - proportionate and least intrusive response appropriate to the risk presented
    - partnership
        - local solutions through services working with their communities
        - communities have a part to play in preventing, detecting and reporting neglect and abuse
    - accountability
        - accountability and transparency in delivering safeguarding
- confidentiality
    - serious case reviews often identify lack of information sharing between agencies as an issue
    - doctors must ensure that they share information about their concerns whilst respecting confidentiality
    - if a person has capacity under the Mental Capacity Act 2005, doctors have no legal authority to make best interests decisions on their behalf
    - patients and their carers need to be informed that their right to confidentiality is not absolute and that information may be shared in some cases where there is a significant risk of harm to others and in cases where it is in the public interest
- presentation
    - potential or actual abuse is not always obvious and can go unnoticed for long periods of time

    - when assessing abuse, doctors should seek to establish the circumstances surrounding the concerns
    - the abused person may have difficulty in reporting the abuse and may be frightened that it will become worse if they report it
    - an abused adult may become withdrawn, unkempt, lose weight and have poor skin care
        - this may be due to illness or neglect
        - it is important to establish whether the person can reach a drink, feed themselves, and ask for help if needed
    - unexplained injuries may be discovered on examination or reported
        - they should be followed up and the cause of the injury clarified
    - the distress caused by abuse may cause the person to have behavioural change, such as becoming withdrawn, aggressive, irritable or emotionally labile
    - if you suspect neglect or abuse, you must act on it and not assume that someone else will
- assessment
    - factors to consider when inquiring about abuse include:
        - the vulnerability of the individual
        - the nature and extent of the abuse
        - the length of time it has been occurring
        - the impact on the individual
        - the risk of repeated or increasingly serious acts
    - managing the conversation
        - make sure the alleged abuser is not present
        - it may be helpful for the potentially abused patient to be accompanied by someone trusted
        - ensure they have appropriate support to express themselves, including an interpreter if needed
        - be clear what will happen with information disclosed
        - establish the facts of the allegation of abuse and acknowledge the impact of the abuse on the victim
- management
    - in all cases of suspected abuse, it is important to assess the risk to the individual and whether there is a need for immediate intervention
        - it is also important to consider risk to other adults, members of the public, or children
    - referral should be done to the safeguarding team

XC4 domestic abuse

- definition
    - ‘any incident of threatening behaviour, violence or abuse between adults who are or have been intimate partners or family members, regardless of gender or sexuality
    - it involves a pattern of abusive and controlling behaviour by which the abuser obtains power over their victim, and it is seen throughout society, regardless of age, race, gender, sexuality, area or social class
    - includes coercive behaviour
    - starts from the age of 16

    - encompasses issues such as forced marriage, female genital mutilation, 'honour' killings and elder abuse
    - can include, but is not limited to:
        - physical
        - psychological
        - sexual
        - financial
        - emotional
    - lifetime risk of domestic violence (from age 16) is 30% in women and 16% in men
- assessment
    - consider asking about domestic abuse in patients presenting with health markers of domestic abuse, including:
        - depression
        - unexplained symptoms/nonspecific symptoms
        - tiredness
        - chronic pain
        - genital injuries
        - sexually transmitted infections
        - self-harm
        - frequent attendance
        - delay between injury and presentation
        - injuries inconsistent with the explanation, or injuries at different stages of healing
- risk factors for domestic violence
    - female gender
    - young age
    - pregnancy
        - 30% of domestic violence starts in pregnancy
    - substance abuse
    - mental ill health
    - chronic illness or disability
    - previous domestic violence or sexual assault
    - cultural factors, e.g.:
        - living in a community which practices female genital mutilation or 'honour' crimes
        - isolation
        - immigration status
        - language barriers
    - leaving a violent partner
    - stalking
- discussion with patient
    - explore your concerns with the patient in a sympathetic, non-judgmental manner
    - ensure they feel safe
    - see them on their own, without their partner or children
    - use an interpreter if needed
    - reasons for reluctance to disclose include:
        - fear of retribution from the perpetrator of the abuse
        - shame or embarrassment

        - fear that their children may be removed from their care
        - fear of an unsympathetic response or not being believed
        - cultural stigma
        - not believing that you can do anything to help
    - indirect questions include:
        - is everything alright at home?
        - are you getting on with your partner?
    - direct questions (HARK)
        - **h**umiliation
            - in the last year, have you been humiliated or emotionally abused in other ways by your partner?
            - does your partner make you feel bad about yourself?
            - do you feel you can do nothing right?
        - **a**fraid
            - in the last year, have you been afraid or your partner or ex-partner?
            - what does your partner do that scares you?
        - **r**ape
            - in the last year have you been raped by your partner or forced to have any kind of sexual activity?
            - do you ever feel you have to have sex when you don't want to?
            - are you ever forced to do anything you are not comfortable with?
        - **k**ick
            - in the last year have you been physically hurt by your partner?
            - does your partner threaten to hurt you?
- signs that children may be living with domestic violence
    - they actively disclose information about it
    - injuries to themselves
    - anxiety
    - depression
    - unexplained illness
    - constant worry about family members and their safety
    - insomnia or nightmares
    - failure to thrive
    - poor achievement at school, poor attendance
    - behavioural difficulties
    - bedwetting
    - self-harm
    - speech and language delays
    - substance abuse
    - missed health appointments
- management
    - ensure that there is no immediate risk to safety for the patient or any children
        - contact police and initiate child protection procedures if needed
    - complete a domestic violence referral (MARAC form)
        - the MARAC (local multi-agency risk assessment conference) may include representatives from:
            - police

                - probation service
                - victim support services
                - schools
                - social care
                - healthcare
                - women's aid/refuge
                - housing department
                - substance abuse services
            - the person suffering abuse may be represented at the conference by an independent domestic violence adviser (IDVA)
        - a safety plan should be provided, with contact information for agencies should the patient not wish to engage at the current time

SAFEGUARDING AND PSYCHO-SOCIAL EMERGENCIES IN CHILDREN

SaP1 self-harm in children and adolescents

- definitions
    - Mental Health Foundation (2003)
        - 'deliberate self-harm is self-harm without suicidal intent, resulting in non-fatal injury'
        - 'attempted suicide is self-harm with intent to take life, resulting in non-fatal injury'
        - 'suicide is self-harm resulting in death'
- background
    - deliberate self-harm is a common precursor to suicide and young people may kill themselves by accident
    - self-harm is a wide range of behaviours that someone does to themselves in a deliberate and usually hidden way
        - it can go on for a long time without being discovered
- triggers for self-harm
    - a wide range of life events can trigger self-harm behaviour, such as:
        - bereavement
        - bullying at school
        - cyberbullying
        - homophobic bullying
        - mental health problems
        - domestic abuse
        - child abuse
        - conflict between child and parents
- types of self-harm
    - signs of distress include:
        - cutting behaviours
        - other forms of self-harm:
            - burning
            - scalding
            - banging

            - hair pulling
        - self-poisoning
        - not looking after their needs properly, emotionally or physically
        - direct injury:
            - scratching
            - cutting
            - burning
            - hitting oneself
            - swallowing or putting things inside oneself
            - staying in an abusive relationship
            - taking risks too easily
            - eating distress (anorexia, bulimia)
            - addiction (e.g. alcohol, drugs)
            - low self-esteem and expressions of hopelessness
- risks
    - an assessment of risk should include:
        - level of planning and intent
        - frequency of thoughts and actions
        - signs of depression
        - signs of substance misuse
        - previous history of self-harm or suicide in the wider family or peer group
        - delusional thoughts and behaviours
        - feeling overwhelmed and without any control or the situation
- issues to explore
    - how long have they felt like this?
    - are they at risk of harm from others?
    - are they worried about something?
    - ask about their health and other problems such as relationship difficulties, abuse and sexual orientation issues
    - what other risk taking behaviour have they been involved in?
    - what have they been doing that helps?
    - what are they doing that stops the self-harming behaviour from getting worse?
    - what can be done at school or at home to help them with this?
    - how are they feeling generally at the moment?
    - what needs to happen for them to feel better?
- NICE guidance on need for hospital treatment
    - triage, assessment and treatment for young people should take place in a separate area of the ED
    - there should be overnight admission to a paediatric or adolescent ward with CAMHS assessment the next day
    - assessment should be undertaken by healthcare practitioners experienced in the field
    - assessment should follow the same principles as for adults who self-harm, but should also include a full assessment of the family, their social situation, family history and child protection issues

- initial management should include advising carers of the need to remove all medications or other means of self-harm available to the child or young person who has self harmed
- any child or young person refusing admission should be reviewed by a senior paediatrician in the ED, and if necessary their management discussed with the on-call Child and Adolescent Psychiatrist

SaP2 concerning presentation

- risk factors for child abuse (NB can happen to ANY child)
  - care giver factors
    - criminal history
    - mental health history
    - substance abuse
    - young/single parent
    - former victim of abuse/neglect
  - child factors
    - young age
    - behavioural problems
    - chronic illness or disability
    - non-biological relationship to carer
    - prematurity/low birth weight
  - family and environmental factors
    - high local unemployment
    - intimate partner violence in the home
    - poverty
    - social isolation
    - lack of social supports
- when to consider NAI
  - the story is vague and lacks detail
  - the story changes each time it's told
  - there is a delay in seeking medical help
  - the history is not compatible with the injury
  - the history is not compatible with the child's developmental stage
  - there are multiple previous attendances with injuries or illnesses
  - there is a history of violence or domestic abuse in the family
  - the child's interaction with carers appears abnormal
- physical abuse
  - can take many forms
    - burns are most common, followed by fractures and bruises
  - when examining, pay attention to:
    - injuries that do not fit the history
    - multiple fractures in various stages of healing, or different types of injury
    - injuries that are likely to be inflicted
    - evidence of poor caretaking
    - sudden onset of altered mental status not attributable to medical illness
    - any bruising in a child that is not yet cruising
    - bruising to the pinna, neck or abdomen
    - injury to the genitalia

- concerning patterns of bruising
  - location
    - buttocks
    - trunk
    - genitals
    - ears
    - back of hands
  - pattern
    - bilateral
    - symmetrical
    - geometric
  - shape
    - bruise resembles shape of an instrument
      - e.g. belt buckle, hand knuckles, spoon
  - colours
    - multiple bruises of various colours in the same area
  - remember that children who cannot cruise cannot bruise
- concerning features of burns
  - 15-25% are thought to be related to abuse
  - patterns that mimic objects
    - hot plates
    - hair straightening irons
    - steam irons
    - cigarettes
  - immersion burns
    - on buttocks, hands and feet
    - look for absence of splashes, which suggests the child was unable to splash around, which they would if it was accidental
- concerning features of fractures
  - inability to match stated mechanism to fracture pattern
- low GCS
  - NAI should be considered in any non-verbal child presenting with a low GCS
  - the classic triad is retinal haemorrhages, subdural haemorrhage and rib fractures but all information is not available in the ED

- sexual abuse
  - children may say that they have been raped, but more commonly signs are subtle and must be looked for
    - e.g. STIs, excessive sexual knowledge, genital trauma
  - it should be routine practice with adolescents to ask about relationships
    - with whom, how old and whether they are sexually active
  - be aware of the possibility of child sexual exploitation
- emotional abuse
  - when an adult harms a child's development by repeatedly treating and speaking to a child in ways that damage the child's ability to feel and express their feelings
  - signs include:
    - parent or guardian constantly criticising the child
    - child shows extremes of behaviour and displays anxiety

        - delayed physical, emotional or intellectual development
        - compulsive lying and stealing
        - displays feelings of worthlessness
        - eating hungrily or hardly at all
        - attention seeking
        - reluctance to go home
        - fearfulness when approached by a person known to them
- neglect
    - commonest form of abuse reported to services
    - the NSPCC reports that 1 in 6 young people are neglected at some point in childhood
    - it encompasses actual and potential harm
    - considerations include:
        - soiled clothes
        - inappropriate clothes for the weather
        - if someone other than a parent brought them in and why
        - whether the parents are supportive of the interventions being done
        - inappropriate interaction between parents
        - comparison to other siblings present
- presentations
    - neglect
        - failure of provision of basic needs
            - frequent or severe infestations (e.g. scabies, head lice)
            - regular inappropriate clothing or footwear
            - evidence of failure to thrive
            - child is persistently smelly or dirty
            - reports of home hygiene poor enough to affect health, inadequate provision of food, unsafe living environment
            - evidence of child abandonment
        - failure to provide adequate supervision
            - injuries suggestive of inadequate supervision
                - e.g. sunburn, ingestion of harmful substance, near drowning, animal bite
            - evidence that the child is being cared for by a person unable to provide adequate care
        - failure to provide access to appropriate medical care or education
            - parents or carers do not give prescribed medication
            - repeated failure to attend appointments
            - persistent failure to engage with immunisations, regular development reviews or screening
            - failure to seek treatment for dental caries
            - failure to seek medical care to the extent that the child's health or wellbeing is compromised
            - unjustified poor attendance at school
    - physical abuse
        - bruises
            - bruises in the shape of an object
                - fingertips, hand, ligature, stick, tooth mark, belt buckle

        - bruising on a non-mobile baby
        - bruising or petechiae in the absence of a medical condition that do not have a suitable explanation
            - multiple bruises
            - bruises of similar shape or size
            - bruises at sites where accidental bruising is unusual
                - face, eyes, ears, safe triangle of neck, inner arms, buttocks, abdomen, groins
            - bruising suggestive of strangulation on the neck
            - bruising on a child not independently mobile due to age or disability
- bites
    - human bites other than those thought to be caused by another young child
    - animal bites (consider neglect)
- lacerations, abrasions and scars
    - on a baby or child not independently mobile
    - multiple lesions
    - symmetrical lesions
    - on areas usually covered by clothing
    - on the eyes, ears or side of the face
    - lesions which look like ligature marks on wrists, ankles or neck
- burns and scalds
    - where the explanation is not consistent with the injury
    - if the child is not independently mobile
    - on areas of skin which would not be expected to come into contact with the hot object
    - where the affected area is shaped like a recognisable object (e.g. iron, cigarette end)
    - pattern suggestive of forced immersion in boiling water
    - consider neglect (lack of supervision)
- cold injuries
    - hypothermia with no explanation
    - cold swollen hands or feet
- fractures
    - fractures of different ages
    - explanation not consistent with the injury
    - occult fracture on x-ray
    - child not independently mobile
- intracranial injuries
    - inconsistent explanation
    - child less than 3 years old
    - associated retinal haemorrhages, rib or limb fractures or other associated injuries
    - multiple subdural haemorrhages
- eye injuries
    - retinal haemorrhages

                - eye injuries in the absence of major accidental trauma or medical cause
            - spinal injuries
                - any spinal injury in the absence of confirmed major accidental trauma
            - other injuries
                - any visceral injury in the absence of confirmed major accidental trauma
                - any oral injury without adequate explanation
                - any unusual or serious injury
                - delay in presentation
    - fabricated or induced illness
        - symptoms or signs only occur in the presence of one person
        - multiple specialists or opinions sought or involved
        - inexplicably poor response to treatment
        - unlikely history of events
        - discrepancy in the clinical picture
        - reporting of new symptoms as soon as previous ones resolve
        - compromise of normal daily activities of the child
    - female genital mutilation
        - statutory duty to report in the UK in girls under 18
    - emotional abuse
        - behaviour of child
            - reported changes in behaviour or emotional state without other cause, e.g.:
                - recurring nightmares with similar themes
                - extreme distress
                - withdrawal of communication
                - becoming withdrawn
                - aggressive behaviour
            - behaviour or emotional state not consistent with age and with no other cause such as medical condition
                - being fearful, withdrawn or having low self-esteem
                - aggressive behaviour
                - body rocking
                - affection-seeking or attention-seeking behaviour
                - over-friendliness to professionals and strangers
                - excessive clinginess
                - inappropriate interpersonal interactions with carer
                - over-obedience
            - extreme or inappropriate emotional responses not due to age or medical conditions
                - temper tantrums in school age children
                - frequent rages with little provocation
                - inconsolable crying
            - dissociative behaviour
            - child has responsibilities which interfere with normal daily activity

                - child responds to medical examination in an unusual or inappropriate way
                - self-harming behaviour
                - inappropriate bedwetting or soiling
                - the child has run away
                - disturbance in eating behaviour
                    - e.g. repeated hoarding, scavenging, hiding or stealing food
                - delayed development
                    - physical, mental, emotional, speech disorders
            - behaviour of parent or carer
                - reporting of bedwetting or punishment for bedwetting despite being told by professionals that it is involuntary
                - rejecting or scapegoating of the child or young person
                - inappropriate expectations
                - inappropriate threats or disciplining
                - using the child to fulfil an adult's needs
                - refusal to allow a child to speak to health professionals on their own
                - unresponsiveness towards a child
                - exposure of a child to frightening events such as domestic abuse
                - failure to promote appropriate socialisation of a child
        - sexual abuse
            - see section below

SaC1 conditions presenting as a symptom of NAI or psychological distress e.g. deliberate self harm, aggression or risk taking behaviour, recurrent abdominal pain, headaches or faints, recurrent attendances in young children

- aggression
    - violence and aggression in children and young people – NICE pathway
        - general principles
            - manage in line with the recommendations for adults, taking into account:
                - the child or young person's level of physical, intellectual, emotional and psychological maturity
                - that the Mental Capacity Act 2005 applies to young people age 16 and over
            - collaborate with those with parental responsibility
            - use safeguarding procedures
            - involve the child or young person in taking decisions about their care wherever possible
        - assessment and initial management
            - assess and manage any underlying mental health problems in line with relevant NICE recommendations
                - including those on antisocial behaviour and conduct disorders in children and young people, attention deficit hyperactivity disorder, psychosis and schizophrenia and autistic spectrum disorder

- identify any history of aggression or aggression trigger factors, including experience of abuse or trauma and previous response to management of violence and aggression
- identify cognitive, language, communication and cultural factors that may increase the risk of violence or aggression in a child or young person
- consider offering children and young people with a history of violence or aggression psychological help to develop greater self-control and techniques for self-soothing
- offer support and age-appropriate interventions (including parent training programmes) in line with NICE recommendations on antisocial behaviour and conduct disorders in children and young people to parents of children and young people whose behaviour is violent or aggressive

▪ de-escalation
- use de-escalation techniques in line with the recommendations for adults, modified for children and young adults and:
  - use calming techniques and distraction
  - offer the child or young person the opportunity to move away from the situation in which the violence or aggression is occurring (e.g. to a quiet room or area)
  - aim to build emotional bridges and maintain a therapeutic relationship

▪ restrictive interventions
- use only if all attempts to defuse the situation have failed and the child or young person becomes aggressive or violent
- when restrictive interventions are used, monitor the child or young person's wellbeing closely and continuously, and ensure their physical and emotional comfort
- do not use punishments, such as removing contact with parents or carers or access to social interaction, withholding nutrition or fluids, or corporal punishment, to enforce compliance
- manual restraint
  - if possible, allocate a member of staff who is the same sex as the child or young person to carry out manual restraint
- mechanical restraint
  - do not use mechanical restraint in children
    - only used when transferring between medium- and high-secure settings and remove at the earliest opportunity (e.g. handcuffs)
- rapid tranquilisation
  - IM lorazepam with dose adjusted according to age and weight
  - if there is only a partial response, check the dose again according to age and weight and consider a further dose

- risk-taking behaviour
  - common types of risk taking behaviour

        - accidents and injury
            - road accidents
                - inexperience
                - driving when tired
                - drink/drug driving
                - carrying passengers
                - mobile phone use whilst driving
            - suicides
            - self-inflicted injury
            - violence and homicide
        - mental disorders and self-harm
        - sexual activity
            - lack of condom use
            - sexually transmitted infections
        - unintended pregnancy
        - tobacco use
            - most adults who smoke begin in adolescence
        - alcohol use
            - toxicity to the developing brain
            - impairment of judgement and lowered inhibitions
            - requirement for medical attention as a consequence
        - drug use
        - nutrition and exercise
            - adoption of unhealthy dietary patterns that persist into adulthood
            - lack of physical activity
                - should aim for 60 minutes/day
            - being overweight or obese
            - eating disorders
    - barriers for adolescents accessing healthcare
        - 5Cs and a D
            - cost (less likely to be an issue in the UK but may include transport cost/time not earning)
            - confidentiality (fear of lack of)
            - compassion (judgemental clinicians)
            - clinical skill (poor communication by physician and lack of evidence based management of common problems)
            - convenience (waiting times, inflexible appointment systems)
            - developmental issues (e.g. embarrassment, poor self-identified needs, low knowledge of services, low health literacy)
    - HEEADSSS assessment
        - **h**ome environment
        - **e**ducation and employment
        - **e**ating and exercise
        - **p**eer-related activities
        - **d**rugs, tobacco and alcohol
        - **s**ex and sexuality
        - **s**uicide, depression and other mental health issues

        - **s**afety from injury, violence, abuse, vaccine preventable infections
    - approach to consultations
        - introduction, welcome, explain process, empower, validate
            - welcome and introduce self
            - validate concerns of the patient (and parent/guardian if present)
            - acknowledge and praise help-seeking and reassure that you can help
            - negotiate time alone during the consultation with the young person
            - avoid asking personal/sensitive questions in front of parents/guardians/partners
            - if friends are present, ask if the young person is comfortable for them to stay and hear personal details
        - explain confidentiality and signpost a preventive care psychosocial assessment
            - discuss confidentiality and its exceptions (risk of harm including suicide/ homicide/ physical abuse/ sexual abuse
            - assess presenting issue, elicit concerns/fears
            - explain the process (history, examination if appropriate, feedback, plan)
            - explain the purpose of questions about personal life and lifestyle (to check if lifestyle behaviours are affecting health and wellbeing)
            - ask permission before asking personal/sensitive questions
            - give permission to decline to answer personal questions
        - conduct a psychosocial risk and protective factor assessment
            - conduct HEEADSSS assessment
            - ask about and comment on what is going well (strengths)
            - make judgement about overall level of psychosocial risk (e.g. low, low-medium but vulnerable, troubled, out of control)
            - provide feedback to the young person on aspects of their life you have assessed as going well and those needing further work; praise engagement in the process
        - make a shared management plan
            - negotiate a short, medium and long term plan
                - likely to only be short-term in the ED
            - assess capacity to consent if they are under 18
            - rehearse what might be discussed with parents/guardians
            - provide education if required about the health issue and ongoing care
            - use preventive care toolkit (motivational interviewing, health promotion)
                - likely to happen in GP/CAMHS
            - referral for counselling and GP review after this
            - instil a sense of hope
- recurrent abdominal pain
    - background
        - defined as at least 3 episodes of pain over at least 3 months
        - affects the child's ability to perform normal activities
        - usually functional (non-organic) but an organic cause is found in 5-10%

        - it is essential to consider and exclude organic causes without overinvestigating
    - epidemiology
        - affects 10-20% of school-age children worldwide
        - patients with organic pathology usually display ‘alarm’ features
        - most common in children aged 5-14
            - rare under the age of 5
        - more common in girls
        - associated with stress
            - children are more likely to have experienced events such as death of family members, domestic violence, harsh punishment from parents, parental job loss and economic stress, hospitalisation, bullying
        - appears unrelated to socioeconomic group
    - associated symptoms
        - children are more likely to have:
            - headache
            - joint pain
            - anorexia
            - vomiting
            - nausea
            - excessive gas
            - altered bowel symptoms
            - anxiety and depression
            - absence from school
    - risk factors for recurrent abdominal pain
        - parental anxiety in the first year of life
        - parents with a gastrointestinal problem
        - history of illness in siblings
        - ADHD
        - being bullied
        - child abuse, including sexual abuse and neglect
    - presentations
        - symptom groups as defined by Rome criteria:
            - childhood IBS (>70%)
            - functional dyspepsia
                - postprandial distress syndrome
                - epigastric pain syndrome
            - abdominal pain
            - functional abdominal pain (FAP)
            - FAP syndrome
    - paediatric IBS
        - at least 4 times a month for at least 2 months:
            - abdominal pain, which at least 25% of the time is:
                - improved with defaecation AND
                - onset is associated in change of stool frequency or form

        - after appropriate medical evaluation the symptoms cannot be attributed to another medical condition
        - there are three types: constipation-predominant, diarrhoea-predominant, mixed or alternating type
        - children often have a sense of incomplete evacuation and sit on the toilet for a long time
    - functional dyspepsia
        - postprandial pain syndrome
            - for at least two months:
                - troublesome postprandial fullness, occurring after ordinary meals, several times per week AND
                - early satiation that prevents finishing a regular meal, several times per week
            - other symptoms may include:
                - upper abdominal bloating or postprandial nausea
                - excessive belching
                - epigastric pain syndrome which may co-exist
        - epigastric pain syndrome
            - at least 4 times a month for at least 2 months
                - intermittent pain or burning localised to the epigastrium, of at least moderate severity
                    - pain is not generalised or localised to other areas
                    - is not relieved by defaecation or passage of flatus
                    - does not fulfil criteria for biliary pain
                - other symptoms may include:
                    - epigastric pain of a burning quality, without a retrosternal component
                    - pain induced or relieved by ingestion of a meal, also occurs whilst fasting
                    - postprandial distress syndrome which may coexist
    - abdominal migraine
        - at least twice in the preceding 12 months, all of the following:
            - paroxysmal episodes of intense, acute periumbilical pain lasting at least an hour
            - intervening periods of normal health, lasting weeks to months
            - the pain interferes with normal activities
            - the pain is associated with two or more of:
                - anorexia
                - nausea
                - vomiting
                - headache
                - photophobia
                - pallor
            - after appropriate medical evaluation the symptoms cannot be attributed to another medical condition
    - functional abdominal pain
        - at least 4 times a month for at least 2 months:

- episodic or continuous abdominal pain
- insufficient criteria for other functional gastrointestinal disorders
- after appropriate medical evaluation the symptoms cannot be attributed to another medical condition

- FAP syndrome
  - at least 4 times a month for 2 months:
    - impairment of normal activities
    - FAP in at least 25% of episodes
    - somatic symptoms such as headache, limb pain, difficulty in sleeping
- presentations suggestive of organic disease
  - the presence of alarm symptoms increases the likelihood of organic disease and should prompt further investigation
  - diagnostic studies are unlikely to be helpful in the absence of alarm symptoms, although re-review is important
  - alarm symptoms:
    - involuntary weight loss
    - falling off growth centiles
    - gastrointestinal blood loss
    - significant vomiting
    - chronic severe diarrhoea
    - unexplained fever
    - persistent right upper or right lower quadrant pain
    - family history of inflammatory bowel disease
    - abnormal physical signs such as:
      - pallor
      - jaundice
      - guarding
      - rebound tenderness
      - altered bowel sounds
      - palpable mass
    - joint inflammation
    - oral and/or perianal lesions
    - skin rashes
    - delayed puberty
    - any features suggestive of child abuse, including sexual abuse and neglect
- typical features of organic vs non-organic causes
  - organic
    - site of pain
      - anywhere
        - particularly flanks and suprapubic area
        - especially note persistent right upper or lower quadrant pain
    - family history (particularly abdominal pain, headache, depression)
      - less likely
      - take note of family history of inflammatory bowel disease
    - psychological factors (particularly anxiety)

        - less likely
    - headache
        - less likely
    - alarm symptoms
        - more likely
            - vomiting
            - chronic severe diarrhoea
            - unexplained fever
            - gastrointestinal blood loss
    - abnormal signs
        - present
    - abnormal growth and/or involuntary weight loss
        - present
    - abnormal investigations (e.g. FBC, ESR, urinalysis)
        - expected
- non-organic causes
    - site of pain
        - usually central and often epigastric
    - family history (particularly abdominal pain, headache, depression)
        - more likely
    - psychological factors (particularly anxiety)
        - anxiety more likely
    - headache
        - more likely
    - alarm symptoms
        - less likely
    - abnormal signs
        - absent
    - abnormal growth and/or involuntary weight loss
        - absent
    - abnormal investigations
        - not found

- assessment
    - history
        - site of pain
        - quality and nature of pain
        - whether pain is relieved by defaecation or not
        - associated symptoms
            - e.g. headache, tiredness, belching, altered bowel habit
        - severity of pain
            - pain scales may be helpful but can be misleading
        - effect of the pain on school attendance, physical activity and daily living
        - beliefs and concern of the child and their parents regarding the source and meaning of the pain and their expectations
        - diet, including any known or suspected allergies or intolerances
        - family history of bowel disorders

- focus on:
    - gastrointestinal symptoms, including bowel habit
    - genitourinary symptoms
    - past medical history
        - including past illnesses, hospital admissions, relevant perinatal and neonatal history
- examination
    - plot height and weight on growth chart
    - check for:
        - anaemia
        - jaundice
        - mouth ulcers
        - skin rash
        - arthritis
    - ask the child to point with one finger to where the pain is worst and most frequently felt
        - most often around the umbilicus in IBS
    - inspect the abdomen for scars, distension
    - perianal appearance
        - prominent perianal skin tags or fistulae suggestive of Crohn's
    - palpation
        - organomegaly
        - tenderness
        - abdominal masses
    - there is typically vague tenderness without guarding or rigidity and normal bowel sounds

- investigations
    - likely not required in the first instance, particularly if no alarm features
    - reassurance and explanation regarding the likely cause of the pain
    - investigations to consider (may be deferrable to primary care after a period of observation if child is well):
        - coeliac screen
        - FBC
        - CRP
        - stool sample for giardia
        - urine microscopy
- differential
    - otherwise well child
        - constipation (may coexist with IBS)
        - coeliac disease
        - lactose intolerance (including transient symptoms after gastroenteritis)
        - giardiasis
        - mesenteric adenitis
        - urinary tract infections
        - mittelschmerz

                - period pain
            - typically presenting with red flags
                - intussusception
                    - uncommon over 18 months
                - GORD
                - Crohn's disease
                - ulcerative colitis
                - urolithiasis
                - pancreatitis
                - Meckel's diverticulum
                - peptic ulcer disease
                - Wilm's tumour
                    - peaks at 3-4 years, rare over 10 years
                - helminth infestation (rare in UK)
                - childhood abdominal sarcomas
                - pregnancy
                - sexually transmitted infection
    - management
        - explanation of diagnosis
            - including that physical pain and symptoms is a normal response to stress
        - explanation of strategies to cope with stress
        - reassurance of no serious underlying disease
        - parents should be advised to reduce concerned responses and focus on distraction instead
            - things that reinforce symptoms should be removed (such as time off school with TV access, being excused from homework)
        - children should attend school regardless of pain
            - schools may need a letter confirming that the pain is real, but non-organic
            - gradual return to school is not advised as it focuses on sickness rather than wellness
        - there is some evidence that probiotics may be helpful
        - later management if required may include biopsychosocial therapies such as hypnotherapy, CBT, yoga exercises and acupuncture
    - prognosis
        - possible risk factors for chronicity
            - presentation under 6 years
            - history of more than 6 months before presentation
            - parental functional problems, stressful life events, sexual abuse
        - children are more likely to have recovered at follow-up if their parents accept a psychological cause for the symptoms
- recurrent headaches
    - background
        - may be primary or secondary
        - the majority are benign and need no investigations
    - history

        - include:
            - frequency
                - and any recent change in frequency
            - when they started
            - description of headache
                - aura
                - location of pain
                - how long it lasts
                - associated symptoms
                    - nausea
                    - vomiting
                    - dizziness
                    - visual disturbance
                    - abdominal pain
                - aggravating and relieving factors
                - any neurological symptoms during the attack
                    - weakness
                    - numbness
                    - loss of sensation
                - medications
                - family history of headache or migraine
    - examination
        - will usually be normal
        - overall growth and development assessment
        - head circumference in younger children
        - signs of potential secondary causes:
            - sinusitis
            - dental problems
            - otitis media
            - TMJ dysfunction
        - blood pressure
        - fundoscopy in older children who will tolerate it
        - neurological examination
            - cranial nerves
            - limb examination
            - gait
            - cerebellar examination
    - red flags
        - signs of raised ICP
            - worse in the morning
            - worse on coughing/straining/leaning forward
        - any focal neurological sign
        - confusion
        - reduced level of consciousness
        - be suspicious of:
            - occipital headache
            - persistent symptoms

- fever
- persistent headaches in children under 4

- migraine
  - two major types
    - migraine with aura
      - transient focal neurological symptoms occur before or during the headache
      - symptoms are typically visual, sensory or speech
    - migraine without aura
      - headache with associated symptoms lasting 4-72 hours
      - often associated with photophobia
      - pain is commonly fronto-temporal and can be unilateral
      - can feel like a throbbing/pulsing pain
      - there may be nausea
  - diagnostic criteria

- at least 5 attacks fulfilling criteria B-D
- headache attacks lasting 4-72 hours (when untreated or unsuccessfully treated)
- headache has at least two of:
  - unilateral location
  - pulsating quality
  - moderate or severe pain intensity
  - aggravation by or causing avoidance of routine physical activity (e.g. walking or climbing stairs)
- during headache at least one of the following:
  - nausea and/or vomiting
  - photophobia and phonophobia
- not better accounted for by another ICHD-3 diagnosis
  - history
    - family history of migraine
    - triggers such as chocolate, MSG, citrus, menstruation

- tension headaches
  - usually described as band-like pain that is persistent but not progressive
  - may be more common in patients undergoing stressful life events such as parental separation or exams
  - use the HEADSSS screen in older children
    - home
    - education
    - activities
    - drinking/drugs
    - sex
    - safety
    - suicide
  - most are managed by reassurance, reducing any stressors and simple analgesia (paracetamol, ibuprofen)
- cluster headaches
  - less common but can occur in children
  - present as unilateral pain in or around the eye or the temporal region

        - can last for up to three hours and can occur up to eight times per day
        - there are often associated unilateral facial signs:
            - conjunctival injection
            - rhinorrhoea
            - lacrimation
            - facial sweating
        - occasionally the child can be treated with triptans for prophylaxis, otherwise they can try simple analgesia (paracetamol, ibuprofen)
    - analgesic headache
        - uncommon in children
        - occur when children take regular daily medication for headache
        - headache should resolve within a few weeks of stopping the medication
    - raised intracranial pressure
        - if red flags present, requires further investigation
            - CT if unwell, possibly admission for MRI if stable
    - trauma
        - always ask as family may not make the connection and mention it
        - may represent intracranial haemorrhage or concussion
    - meningitis
        - the child is often unwell with irritability, photophobia and neck stiffness
        - children may have meningitis without the classical signs so the diagnosis should be considered in children with irritability and particularly reduced consciousness
    - management
        - simple analgesia with paracetamol and ibuprofen is well-tolerated and effective in most children, especially for migraines
        - anti-emetics can be considered if the child has a lot of nausea and vomiting with migraine (ondansetron or prochlorperazine)
        - triptans (e.g. intranasal sumatriptan) can be used for migraine as a stat dose in the ED in children over 12
        - more serious causes and red flags need to be investigated as appropriate
        - consider the social aspects
            - ask about family stressors, school progress and friends
                - identifying stress and conflict can help to reduce headaches
        - identifying triggers can allow avoidance (e.g. caffeine, chocolate, extreme tiredness, missing meals)
        - parents of children with recurrent headaches should be encouraged to keep a symptom and food diary
- recurrent faints
    - background
        - syncope is a sudden and transient loss of consciousness with loss of postural tone from which recovery is spontaneous and complete
        - 15% of children have a syncopal event, the majority of which are benign
        - the differential list is huge, and the role of the ED is to detect the serious causes without overinvestigating the benign causes
    - classification of syncope
        - neurally mediated

        - most common
    - cardiovascular causes
        - potentially fatal
    - other
        - epilepsy and psychogenic
- causes
    - autonomic
        - vasovagal syncope (also called neurocardiogenic)
        - orthostatic hypotension
        - postural orthostatic tachycardia syndrome
        - breath-holding spells
    - cardiac
        - brady/tachyarrhythmia
        - long QT syndrome
        - Brugada syndrome
        - Wolff-Parkinson-White syndrome
        - structural abnormalities (e.g. aortic stenosis, hypertrophic cardiomyopathy)
    - other
        - functional disorder
        - hypoglycaemia
        - seizure
        - migraine
        - anaemia
        - narcolepsy
        - toxic exposure
- history
    - precipitating events
        - including preceding symptoms and the position in which the syncope occurred
        - in syncope:
            - sudden or prolonged standing
            - painful or emotional stimulus
            - palpitations
    - period of unconsciousness
        - usually seconds in syncope
    - incontinence
        - usually absent in syncope
    - confusion on waking
        - absent in syncope
    - tonic-clonic movements
        - occasionally and briefly occur in syncope
    - red flags for potential cardiac causes
        - lack of prodrome
        - palpitations or chest pain
        - exercise-induced
        - past cardiac syncope

                - family history of early cardiac death, arrythmia or sudden death
        - investigations
            - observations
                - including orthostatic BP and heart rate
            - baseline ECG
            - complete cardiac and neurological examination
            - blood glucose if seen shortly after the event
            - pregnancy test if applicable
- recurrent attendance in young children
    - indicators that should prompt consideration of abuse include:
        - frequent attendance or unusual patterns of attendance to healthcare services, including frequent injury
        - change to the child's behaviour or emotional state
        - injury with features of maltreatment
        - evidence of sexual activity
        - harmful interactions between child and carer
        - appearance of neglect
        - failure to access medical care appropriately
            - including non-attendance for routine immunisations, delay in presentation
        - unsuitable explanations
    - consider presence of risk factors for child abuse
        - previous history of child maltreatment in the family
            - health visitors and social workers may have useful information
        - domestic violence
            - also domestic/marital conflict and history of violent offending in the family
        - mental health disorders, learning disability, physical illness or disability in the carers
        - drug or alcohol abuse in the carers – especially if unstable or chaotic drug misuse
        - housing or financial problems
        - disability or long-term chronic illness in the child
        - single parents, especially if immature or unsupported
        - history of animal/pet maltreatment
        - children in the care system
        - some children are vulnerable to being 'lost' by the system
            - e.g. where families are homeless or asylum seekers, or where children are carers or young offenders

SaC2 roles of other systems in protecting children e.g. Social Services, the Child Protection Plan, Police Child Protection and Domestic Violence Units, SureStart, Childline, Health Visitors, School Nurses

- named professionals and child protection leads
    - provide advice within the hospital
- police
    - may enter premises and remove a child to a place of safety for 72 hours

    - have child abuse investigation units, which normally take responsibility for investigating child abuse cases
- social workers (local authority social services)
    - all local authorities have a social services officer permanently on call (including out of hours) with access to the child protection register
        - the officer can take referrals if there are concerns about a child
    - the local authority has responsibility for the safety and welfare of children
- NSPCC
    - a voluntary organisation authorised to initiate child protection proceedings
    - has a national child protection helpline and a children's helpline
- local safeguarding children's board (LSCB)
    - each local authority has a statutory responsibility to set up an LSCB under the 2004 Children's Act
    - it has overall responsibility for deciding how the relevant organisations will work together to safeguard children in its area
    - it develops local safeguarding policies and procedures, and monitors and coordinates them
    - they exist in England and Wales
- child protection case conferences
    - held if the child is at risk of significant harm so that all relevant professionals can share information, identify risks and outline what needs to be done to protect the child
    - people involved in a case conference include:
        - agencies such as social service, the police and health services
        - people who have the most involvement working with the child and family (e.g. school, family GP)
        - family members
        - the child, where appropriate
    - considerations include:
        - background information about the family
        - findings from the child protection investigation
        - ongoing assessments
- child protection plan
    - sets out:
        - how social workers will check on the child's welfare
        - what changes are needed to reduce the risk to the child
        - what support will be offered to the family
    - a core group is responsible for making sure that the child protection plan is supporting the child effectively on an ongoing basis
        - members of the group include the person nominated as the Lead Professional, the child and their parents/carers
        - they report about it at child protection case conference meetings
        - the core group should be set up within 15 days of the initial child protection case conference
- Surestart
    - centres that give help and advice on child and family health, parenting, money, training and employment

    - some centres also provide early learning and day care for pre-school children
- Childline
    - a service run by the NSPCC
    - provides telephone counselling services
    - can also be contacted online and via email
    - free and confidential
    - can also provide sign language interpretation
- health visitors
    - registered nurses/midwives who have additional training in community public health nursing
    - take over from the midwife at day 10 – most will have arranged to meet the family during the last few weeks of pregnancy
    - first point of contact for families with questions around feeding and development
    - regulated by the NMC
- school nurses
    - qualified and registered nurses
    - many have additional experience, training and qualifications to become specialist community public health nurses
    - work across education and health to provide a link between school, home and the community
    - work with families and young people from five to nineteen
    - usually linked to a school or group of schools
    - usually the first point of contact for supporting teachers and children in school with minor health or developmental problems
    - in most areas they run clinics for primary nocturnal enuresis
    - regulated by the NMC

SaC3 Mental illness in childhood including depression, anxiety, OCD, bipolar and schizophrenia

- depression
    - background
        - childhood and adolescent depression often recurs and continues episodically into adulthood
        - prevalence estimated to be 1% in prepubertal children and 3% in postpubertal young people
        - twice as common in adolescent females as males
    - risk factors
        - family discord
        - bullying
        - physical, sexual or emotional abuse
        - history of parental depression
        - ethnic and cultural factors
        - homelessness
        - refugee status
        - living in institutional settings
    - presentation
        - the diagnosis is often missed
        - often presents with somatic symptoms

        - may also have features of anxiety
    - sometimes only presents as poor functioning at school, socially or at home
    - may masquerade as bad behaviour, particularly in boys
    - mood is more variable than in adults and mood swings are common
    - children may be able to enjoy some aspects of their life and this should not preclude the diagnosis of depression
    - features as seen in adults
        - low mood
        - loss of interest
        - socially withdrawn
        - poor self-esteem
        - psychomotor impairment
        - tearfulness
        - guilt
        - anxiety
        - lack of enjoyment in anything
    - features common in childhood
        - running away from home
        - separation anxiety and possibly school refusal
        - complaints of boredom
        - poor school performance
        - antisocial behaviour
        - insomnia
            - often initial and middle rather than early morning wakening
        - hypersomnia
        - eating increased or decreased, particularly if associated with weight change
    - young primary school children may present with sadness and helplessness, slightly older children with feelings of being unloved and unfairly treated; guilt and despair may predominate in teenagers
    - consider the possibility of concealed contributory factors (e.g. past child abuse, bullying)
    - parents may not always be aware of depression in their children
    - assessment is often difficult and questions may be answered with silence or a shrug
    - adolescents with conduct disorders can be manipulative and difficult to assess
    - always ask about suicidal ideation and thoughts of self-harm
    - particular attention should be paid to:
        - confidentiality
        - the young person's consent
        - parental consent
        - child protection
        - use of the Mental Health Act in young people
        - use of the Mental Capacity Act in young people
        - use of the Children Act
- management

        - often based in secondary care
        - reasons for CAMHS referral:
            - depression where one or more family members (parents or children) have multiple risk histories for depression
            - mild depression in those who have not responded to interventions in primary are after 2-3 months
            - moderate or severe depression (including psychotic depression)
            - signs of a recurrence of depression in those who have recovered from previous moderate or severe depression
            - unexplained self-neglect of at least one month's duration that could be harmful to the child/young person's physical health
            - active suicidal ideas or plans
                - high recurrent risk of self-harm or suicide
            - young person or parent/carer request referral
        - social interventions
            - addressing any sources of distress (e.g. bullying) and removing opportunities for self-harm (e.g. paracetamol at home)
        - psychological interventions
            - CBT for young people aged 5-18 with mild depression continuing after two weeks of watchful waiting
                - may be individual, group or family therapy
        - medication
            - should only be used in conjunction with concurrent psychological therapy
            - fluoxetine is the only medication for which trials show the benefits outweigh the risks
        - electroconvulsive therapy
            - used extremely rarely in young people 12-18 years
    - prognosis
        - about 10% recover spontaneously within about 3 months
        - 50% remain clinically depressed at 12 months and 20-30% at two years
        - about 30% have recurrences within 5 years and many develop episodes as adults
        - worse prognosis with:
            - female sex
            - increased guilt
            - previous episodes of depression
            - parental psychopathology
- anxiety
    - background
        - most common mental health disorder experienced by children and young people
        - only a minority access any support and very few receive evidence based treatment
        - barriers to seeking support include:
            - difficulties differentiating between normal and problematic levels of anxiety

                - uncertainty about when and where to seek help
                - stigma-related concerns
                - limited available support
        - identification
            - excessive fear, worry or anxiety – worry may be about a range of things or a specific thing
            - signs
                - physical symptoms
                    - abdominal pain
                    - nausea
                - related behaviours
                    - avoiding anxiety-provoking situations
                    - anger outbursts
                - comorbid problems
                    - depression
                    - autism
                    - ADHD
                    - alcohol or substance abuse
                - increased risk for self-harm behaviour and suicidal ideation
            - impact on function
                - interference with:
                    - school
                    - social life
                    - mood
                    - sleep
                    - behaviour
                    - family life
            - talk to the child/young person and their family separately
            - consider self- and parent-reported questionnaires
        - management
            - provide information about self-help resources and sources of support
            - discuss whether it would be helpful to liaise with others (e.g. school staff)
            - support the patient to face their fears
            - support families with problem solving
            - GP can refer to evidence-based interventions - CBT
- OCD
    - background
        - an impairing condition associated with a specific set of distressing symptoms incorporating repetitive, intrusive thoughts (obsessions) and distressing, time consuming rituals (compulsions)
        - estimated prevalence up to 4% in children and young people
        - if untreated, symptoms wax and wane but typically follow a chronic course with marked functional impairment across many domains
        - aetiology is unknown, although there is likely to be a genetic element with significant environmental influence
        - there are group who develop sudden onset OCD and/or tics after streptococcal infection

- known as PANDAS (paediatric autoimmune neuropsychiatric disorders associated with streptococcus) and more recently PANS (paediatric acute-onset neuropsychiatric syndrome)
- they tend to have more widespread neuropsychiatric difficulties, including enuresis, deterioration in handwriting, impulsivity
- the exact mechanism is unknown, but it seems to respond to usual therapies – effectiveness of prophylactic antibiotics has not been demonstrated

    - diagnosis
        - diagnostic criteria are similar to those in adults
            - children may however have less insight into the irrationality of their obsessions and compulsions
            - it is also important to distinguish true compulsions from normal ritualised behaviour (the behaviour must be distressing and/or impairing to be pathological)
        - ICD-10 diagnostic criteria

- either obsessions or compulsions or both present on most days for a period of 2 weeks
- obsessions (unwanted ideas, images or impulses that repeatedly enter a person's mind) and compulsions (repetitive stereotyped behaviours or mental acts driven by rules that must be applied rigidly) share the following features:
    - patient is aware that these originate from their own mind
    - they are repetitive, unpleasant and distressing to the patient; at least one is perceived as excessive or unreasonable
    - at least one is resisted unsuccessfully, even though others may be present
    - thought of carrying out the obsession or compulsion is not intrinsically pleasurable
- the symptoms must be disabling; even young children will have some insight into the senselessness of the thoughts and behaviours
    - frequently goes undetected for years before diagnosis
        - may present very differently between patients
    - the majority can be identified using a six question screening tool (the Short OCD Screener – recommended by NICE)
        - it is not a diagnostic tool and further investigation is required for those who screen positive
        - Short Obsessive Compulsive Disorder Screener
- do you wash or clean a lot?
- do you check things a lot?
- is there any thought that keeps bothering you that you would like to get rid of but cannot?
- do your daily activities take a long time to finish (e.g. getting ready for school)?
- are you concerned about putting things in a special order or are you very upset by mess?
- do these problems trouble you?
    - differential
        - autistic spectrum disorder
        - tic disorder
        - psychosis

- management
  - CBT is first line
    - 12-20 sessions involving the young person gradually confronting their fears and refraining from carrying out compulsions
    - they are encouraged to wait until their anxiety settles naturally
    - tasks are graded
  - SSRIs are second line
    - sertraline is recommended in the UK as having the best side effect profile
      - a second SSRI may be tried in treatment failure

- bipolar
  - background
    - characterised by alternating periods of mania, depression and normal mood, each lasting weeks to months at a time
    - typically begins during mid-adolescence into the mid-20s
    - rare in children
      - children with intense, unstable moods are more likely to be given a diagnosis of disruptive mood dysregulation disorder and most progress to a depressive rather than bipolar disorder
    - aetiology is likely to be genetic, exacerbated of mimicked by certain drugs (cocaine, amphetamines, certain antidepressants) or environmental toxins (e.g. lead)
      - certain disorders such as thyroid disorders can cause similar symptoms
  - presentation
    - characterised by recurrent episodes of elevated mood (mania or hypomania)
      - in adolescents, mood may be very positive or hyperirritable
      - speech is rapid and pressured, sleep is decreased and self-esteem is inflated
      - mania may reach psychotic proportions
      - judgment may be severely impaired and adolescents may engage in risky behaviours (e.g. promiscuous sex, dangerous driving)
    - alternating depressive episodes can be more frequent
    - prepubertal children may experience dramatic moods but the duration is much shorter (only lasting a few moments)
    - onset is characteristically insidious
      - children typically have a history of always being temperamental and difficult to manage
  - diagnosis
    - clinical evaluation
    - testing for toxicological causes
    - ruling out medical causes
      - e.g. thyroid disorders, brain infections, brain tumours
  - prognosis
    - varies but worsens with each recurrence
    - factors increasing risk of recurrence include:

                - early age of onset
                - severity
                - family psychopathology
                - lack of and/or poor adherence to treatment
        - treatment
            - mania and depression are generally managed with second generation antipsychotics such as aripiprazole, risperidone, plus an SSRI for depression
            - mood stabilisers such as lithium may then be introduced
            - psychotherapy is also important
- schizophrenia
    - NICE guidelines
        - first episode of psychosis
            - comprehensive multidisciplinary assessment
                - psychiatric
                    - mental health problems
                    - risk of harm to self or others
                    - alcohol consumption
                    - prescribed and non-prescribed drug history
                - medical
                    - medical history
                    - full physical examination
                        - identify physical illness including organic brain disorder
                - psychological and psychosocial
                    - social networks
                    - relationships
                    - history of trauma
                - developmental
                    - social, cognitive and motor development skills
                    - coexisting neurodevelopmental conditions
                - physical health and wellbeing
                    - weight
                    - height
                    - smoking
                    - diet
                    - exercise
                    - sexual health
                - social
                    - accommodation
                    - culture and ethnicity
                    - leisure activities and recreation
                    - carer responsibilities
                - educational and occupational
                    - attendance at school or college
                    - educational attainment
                    - employment
                    - functional activity

        - economic
            - family's economic status
- crisis plan
    - the patient should have a crisis plan which includes:
        - possible early warning signs of a crisis and coping strategies
        - support available to help prevent hospitalisation
        - where the child or young person would like to be admitted in the event of hospitalisation
        - definitions of the roles of primary and secondary care professionals and the degree to which parents or carers are involved
        - information about 24 hour access to services
        - the names of key clinical contacts
- treatment options for first episode psychosis
    - oral antipsychotic medication and psychological interventions
    - benefits and side effects of medications should have been discussed with the young person, including:
        - metabolic (e.g. weight gain, diabetes)
        - extrapyramidal (akathisia, dyskinesia, dystonia)
        - cardiovascular (e.g. prolonged QT)
        - hormonal (e.g. increased plasma prolactin)
        - other (e.g. unpleasant subjective experiences)
- ongoing monitoring
    - efficacy, including changes in symptoms and behaviour
    - side effects of treatment
        - taking into account overlap between medication side effects and symptoms of schizophrenia
    - emergence of movement disorders
    - weight
    - height
    - waist circumference
    - pulse and blood pressure
    - fasting blood glucose, HbA1c, blood lipid and prolactin levels
    - adherence
    - physical health
- rapid tranquilisation and restraint
    - caution with high potency antipsychotic medication such as haloperidol
        - particularly in those who have not taken antipsychotics before
        - there is an increased risk of dystonic reactions
    - the child or young person should be offered an opportunity to discuss their experience afterwards

SaC4 sexual abuse

- consider sexual abuse if:

    - there are genital or anal symptoms (bleeding or discharge) or recurrent dysuria without a suitable explanation or medical cause
    - there are foreign bodies in the vagina or anus
    - a gaping anus is observed during an examination (without a medical explanation such as a neurological disorder or severe constipation)
    - hepatitis B or anogenital warts in a child under 13 (unless there is clear evidence of transmission during birth or blood transmission, or non-sexual transmission)
    - a child aged 13-15 is pregnant
    - sexually transmitted infection or pregnancy in a young person aged 16-17 if there is any evidence that the sex was non-consensual or that the young person was being exploited, or that there is a clear difference in power/mental capacity between the two
    - there are behaviours such as self-harm, running away or secondary bedwetting
- suspect sexual abuse if:
    - a child presents with a genital, anal or perianal injury in the absence of a suitable explanation
    - there are persistent or recurrent genital or anal symptoms (bleeding or discharge) associated with behavioural or emotional change
    - a child presents with an anal fissure (unless there is a medical reason such as constipation or Crohn's)
    - a child aged under 13 is pregnant
        - sexual activity with a child under 13 is, by law, sexual abuse – consent is irrelevant at this age
    - a child under 13 presents with an STI (other than proven vertical transmission during pregnancy or childbirth, or blood contamination)
    - a child aged 13-15 presents with an STI other than where the circumstances above are proven or there has been consensual sex with a peer
    - there is evidence of sexualised behaviour in a prepubertal child
- assessment
    - examination of the genitalia should only be performed by an expert
        - an intimate exam should only be undertaken if there is an urgent medical need to do so
    - use open questions and record all answers verbatim
    - a routine general examination can be performed to check general health
        - a forensic examination must only be performed if the physician has the training and facilities to do so
    - the police or SARC will be able to arrange a specialist examination
        - this will include forensic examination and assessment of the need for contraception and STI assessment
        - ideally the forensic exam should be within 24 hours of the event (up to 72 hours is acceptable)
        - depending on the type of assault, DNA can be gathered from 12 hours to 7 days after the event

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